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CLINIC OF DR S W BANDLER

POST GRADUATE HOSPITAL

STERILITY IN WOMEN, WITH ESPECIAL REFERENCE
TO ENDOCRINE TREATMENT OF THE SAME

Pituitrin in Obstetrics Why is Gynecology Becoming More Medical? Facts from Practical Experience which Tend to Prove the Value of Endocrine Therapy

January 24, 1919

THE topic this morning is sterility in women. As the endocrines bear such an important relation to this question it is essential to spend a few moments discussing the physiology of the endocrines before coming to the main subject.

Most of you have heard me speak of some of these points before in connection with numerous clinical cases, and so you will have to take the old with the new. On the chart shown on page 922 is a short outline which we will use as an introduction to the subject of the endocrines as they concern the gynecologist and the obstetrician.

The human body is managed by the endocrine glands of the body. You have an automobile, and it is run by gasoline of one kind. In spite of the fact that it has a competent mechanism, if you are short of gasoline or have a poor quality of gasoline, that invalidates the ~~value~~ of the automobile. But in the human body you have many kinds of gasoline given off by many glands, each gland producing many secretions. The particular division of these secretions we do not yet understand, but

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CHART No I

Development Pelvis — Chlorosis — Anomalies of menstruation — Neurotic symptoms—Tachycardias

Ovary Thyroid Pituitary Menstruation (a crisis) Amenorrhea—Relative Amenorrhea—Dysmenorrhea—(Marriage)

Premenstrual phenomena { Congenital
Acquired } Thirteen times a year upset

Pregnancy (amenorrhea) Toxemia—generally slight—Severe type Action of corpus luteum.

Labor (a crisis) Pituitrin—Forceps

Postpartum asthenia Lactation atrophy—often starvation or *gland exhaustion*

(Mammary Extract)

Sterility Curet?

Repeated abortion Wassermann

Hyperthyroidism Inter glandular upset — Reflex — Hysteria(?) — Neurasthenia(?)

Menopause Flushes { Excited
Depressed } Psychoses.

Postpartum psychoses Puerperal mania

Dementia praecox

each gland and its special components has definite specific action, and every individual from the time he is born until the time he dies is under the influence of these many different kinds of elements—some of them having to do with the development of the bones and teeth, some with the development of the body and nervous system, some with the development of the mind, etc., and, later on—with the introduction of the sex factors—with reproduction Still later on these elements have to do with the preservation of these structures and functions which constitute the body and mind, and if the gasoline elements which are given off by these glands become under- or overactive you have a disturbance of the specific functions these component parts are supposed to perform, and as these glands are dependent upon each other the upset of one disturbs the rhythmic action of the others, so that a woman during her development and maturity keeps in action as her glands keep in normal action, and as she approaches the climacterium and, later on, the years of senility, her glands change and her activities change, so that

if she lives long enough she is pretty nearly back to where her body function started in the early years

We must not forget that the pituitary and thyroid have much to do with the development of the body and bones, as may be seen from cretinism. The development of infantilism is traced in many cases to the lack of proper developmental stimuli by the thyroid or pituitary glands. The development of the *status lymphaticus* shows the importance of the thymus and its associated lymphatic activities. The thymus is supposed to decrease at a time when the gonadal function develops. Hence, among its functions is supposed to be that of inhibiting a too early development of the sex glands. Now, we have a different body development in the male and in the female, and we explain that by the fact that the secretions of the testes so act on the other glands of the body, especially the anterior pituitary and adrenal cortex, as to increase their activity and give a heavier bony framework, larger hands, and larger stature, while the ovaries, not affecting the other glands to the same extent, are accompanied by a lesser stimulation of the anterior lobe of the pituitary and other glands controlling the development of bone, and the result is a smaller stature, smaller hands, etc. Hence the difference in shape and character of the male and female pelvis. *The anterior lobe of the pituitary acts more specifically in the male than in the female, the posterior acts more characteristically in the female than in the male.*

Consider for a moment the question of chlorosis, which is observed exclusively in the girl after the establishment of puberty, and you will see that the etiology of this blood state has been referred to a weak spot in the blood forming function in which the ovary is supposed to play a part. Therefore many of these cases have been benefited by the administration of ovarian extract. Others have been markedly benefited by ovarian extract to which are added iron and arsenic. As a result I have adopted the rule that when I want to increase the activity of the ovarian extract I frequently add iron and arsenic, and I usually add ovarian extract to iron and arsenic when given for any form of anemia unless the patient is suffering from menorrhagia, in which

case the ovarian extract is omitted and an endocrine extract, such as mammary or thymus or anterior pituitary, which diminish menstruation, is added

We now come to the stage of puberty, the time when the girl first menstruates, and in your experience you will notice numerous anomalies of menstruation that which comes on slowly or late, that which comes on at irregular periods, during any of which you may have slight or great degrees of tachycardia, you will observe many cases of menorrhagia, or you will note major or minor forms of neurotic symptoms, so that many of these girls have to be taken out of school. They are nervous and irritable, on edge, cannot concentrate, cannot do their work, and many are filled with peculiar thoughts and obsessions, not infrequently taking on a sexual character. These anomalies of menstruation, these tachycardias, these psychic symptoms are due to the fact that the ovary, in asserting itself as a new and powerful member of the endocrine family, upsets the other members and relationships, until finally, in the vast majority of cases, harmony is established. If it is established easily and quickly, the girl menstruates regularly and there are no annoying symptoms. If harmony be established slowly and with difficulty, you have more of the above-mentioned annoyances. If no permanent smooth action is established, you have a condition where every menstruation acts badly on the system, so that we must speak not only of dysmenorrhea in the old accepted meaning of the term, but instead speak of *constitutional dysmenorrhea*. It is the same as when a man marries, sometimes the new wife fits into the new family relationships with harmony, sometimes not, and just as these families react differently to the introduction of the new member, so the endocrines react to the introduction of the ovarian element.

What do these neurotic symptoms at this developmental stage mean to us?

They mean that hundreds of girls have, from fifteen to nineteen or twenty years of age, various forms of physical and psychic phenomena which, though they smooth themselves out in the vast majority of cases, and eventually allow the possessors

to become normal, suggest at the time *minor* forms of those psychoses which are not so rare during the developmental age. Dementia *præcox*, as you know, is a condition affecting the growing boy or girl at the developmental age. It is in many instances an hereditary condition, and in the large majority of cases it is incurable. What can be made responsible for this condition except an interglandular upset? What makes such a boy or girl develop in a fairly normal way up to a certain stage and then show abnormality, eventually leading on to a well-defined form of dementia *præcox*?

These various points are suggestions, but they mean that every girl or boy is a potential dementia *præcox*. Practically every person is a potential manic-depressive. We all have our periods of elation and of depression, with cause and without cause, but our endocrine relationship preserves a tolerable balance, but if this endocrine upset is pronounced and manifests its activity or its influence mainly on the nervous system, you may have a developmental psychosis. This can be explained on no other basis than endocrine instability and endocrine upset.

Take the example of postpartum mania, where the patient goes out of her mind. What does that mean? That individual may have been perfectly normal all her life. As a result of having a baby, she develops a mania from which she may recover or may not. The trouble does not originate in the brain, which has acted normally all her life, but some gland crisis or glandular anomaly resulting from the introduction and subsequent removal of the placental secretion has so disturbed her cerebral activity that she is like one poisoned by an infectious disease. These cases, however, usually recover. There is, because of some change in the endocrine relationship, a toxic or irritating action on certain brain centers or functions.

We now come to menstruation, a period in which the ovary, pituitary, and thyroid are concerned. At every menstruation these three act or react, and according to the harmonious action of the three you have either a normal menstruation or menorrhagia, a relative amenorrhea, or varying degrees of actual amenorrhea.

Now, the glands which preside especially over menstruation are the ovaries. Without ovaries no menstruation takes place. The ovaries are more or less under the domination of other glands of the body. The thyroid has an important part in preserving and aiding the normal action of the ovary and uterus. The pituitary gland is intimately connected with the genital apparatus. As you know, an affection of the posterior lobe of the pituitary gland, whereby its secretion is diminished, is associated with progressive gain in weight, and a progressive diminution of menstruation, and an eventual greater or lesser degree of atrophy of the uterus and ovaries. On the other hand, if the secretion of the posterior lobe be increased, we have the opposite condition, excessive menstruation and the development of fibromata and myomata, and the so-called fibrosis uteri.

Now in every menstruation the thyroid reacts to the menstrual stimulus and the posterior lobe of the pituitary undoubtedly plays its part. If the posterior lobe of the pituitary reacts excessively during the menstrual stimulus initiated by the ovary, we get a dysmenorrhea, and I believe that many of the dysmenorrheas are due to excessive action of the posterior pituitary lobe during this monthly crisis. Nothing shows the nerve reaction of a patient more than the way she suffers physically or mentally or nervously before menstruation comes on.

Before each monthly crisis the ovarian secretion produces a gradually increasing congestion or hyperemia in the endometrium, but with this is associated a hyperemia of most of the mucous membranes of the body. The mucous membrane of the nose, the larynx, the gastric mucosa, etc., take part in this general hyperemia a week or ten days before menstruation. The thyroid and the posterior lobe react to this ovarian stimulation, hence a patient's interglandular relation is upset in many cases. Gynecologists have an advantage in this respect, that by taking a thorough history of the premenstrual and menstrual phenomena of patients, we have a very, very important index as to the patient's glandular or interglandular stability. Many patients are made aware by no definite symptoms whatsoever that menstruation is coming on. Others feel a fulness in the breasts or a

pain or discomfort in the pelvic region, others have marked discomfort a few days before menstruation, many women are very irritable or are depressed in this premenstrual stage. Some are irritable almost to the degree of slight mania, others cry and show bursts of temper, others have varying degrees of unusual sexual or psychic manifestation. Some have decided headaches, with or without nausea or vomiting. Such an experience reproduced every month not only indicates lack of nerve resistance to these premenstrual stimuli, but profoundly affects the patient more or less disagreeably and more or less permanently.

The manner in which a patient reacts to the premenstrual stage gives me a most valuable index, and I do not doubt its meaning. It is to be expected that in those patients whose glands do not act harmoniously at this period we may take for granted a latent instability of the interglandular relationship, and if you watch your patients closely at the premenstrual phase you will then see them show baby or minor forms of psychic disturbances which markedly suggest the psychoses which we speak of, when of the major type, as *dementia præcox* or manic-depressive insanity, or the various forms of mania and mental aberration.

To conclude this part of the discussion, we may say the less a patient suffers constitutionally before every menstruation, the more normal her menstruation is in amount and character, the less pain she suffers during menstruation, the more normal we may consider that individual's endocrine system. Every menorrhagia, every metrorrhagia, every relative amenorrhea, every greater degree of actual amenorrhea must first be considered from the standpoint of the endocrines. Of course there are many other causes of menorrhagia and metrorrhagia, but these, in turn, must be diagnosed according to the bimanual findings and according to the history.

Why is pregnancy characterized by amenorrhea?

When the fecundated ovum settles in the intra-uterine lining it nests and embeds in the decidua. As it grows, the outer layer of the ovum, which is called the trophoblast, invades the surrounding tissue and digests it by an enzyme action. Cells

from the trophoblast are given off immediately into the circulation, and this giving off continues as the ovum grows and chorionic villi develop and as the subsequent placenta comes into being. This secretion produced by the outer layer of cells and the subsequent chorionic villi and the subsequent placenta has the power to nullify the menstrual stimulus initiated by the ovary and aided by the thyroid and the pituitary. During the entire period of pregnancy the ovaries, the thyroid, the pituitary, the adrenals, and other protective glands of the body are fighting against that new secretion introduced by the growing ovum.

The next evidence of the trophoblast secretion which we have is the nausea and vomiting that pregnant patients complain of. If the protective glands adapt themselves to this new substance, the nausea and vomiting are slight. If they do not adapt themselves readily or quickly, then the nausea and vomiting are more pronounced, and later on in the pregnancy we have the various degrees of toxemia and finally eclampsia—all of which are undoubtedly due to the fact that protective glands do not give these patients the immunity which the vast majority of patients do gain in the course of a few weeks.

When a child develops scarlet fever or diphtheria it is because the adrenals or the pituitary gland, etc., of that child do not render it immune. A weak protective glandular resistance is an invitation to infection. Not long ago I delivered a patient, and on that corridor another patient, an adult, developed diphtheria. What was to be done? Of course we removed the baby from the corridor, and the first thing which we did was to make the Schick test, and as the baby proved to be immune we had no further worry so far as the child was concerned. Why is one child shown to be immune by this test and another child not? This is explained only on the basis of interglandular relationship in the one case which gives immunity, and a lack of that sufficient power in the next child which does not grant immunity. The same holds good in the present epidemic of influenza and influenza pneumonia. It seems to attack persons in the prime of life, in the period when the gonad apparatus is supposedly most active. The epidemic is not so severe in children where the gonad

apparatus is not so developed, nor in older people where the gonadal apparatus has disappeared as a functioning process

Influenza, especially the severe cases, represents a systemic infection, and the poison seems to have a predilection for the uterine mucosa, and many pregnant patients stain or miscarry, and many patients instead of going to full term have a premature labor. Many patients who are not pregnant, within a week after the infection, have an increased menstruation, anticipating the normal one by several days. It must be remembered that the uterine lining is a lymphoid tissue, with the exception of its glands and epithelial cells, resembling the tonsils. The toxic cases in the present epidemic of influenza represent probably an invasion past the lymphoid and lymphatic system when the thymic, colym phatic system and the chromaffin systems fail to give sufficient protection. After invasion of the system, the subsequent course shows that the glands whose function it is to take care of the genital tract are especially affected by the toxin developed by influenza. When a pregnant patient, especially one near full term, is attacked by this pneumonia epidemic her interglandular apparatus (pituitary, adrenal, etc.), already weakened by fighting against placental aggression, is especially liable to injury, and the power of resistance granted by these glands is naturally less than in the normal individual.

Now just a few words about the use of corpus luteum, by mouth or by hypodermic, for the nausea and vomiting of pregnancy. Theoretically speaking, it ought to be very valuable to us, since true corpus luteum is only formed when pregnancy takes place. However, one cannot rely on it as a specific. It may help in some cases, but in routine use it has failed to have any definite value. The average person recovers with mild medication, or after the use of the Murphy drip (2 per cent. sod. bicarb., 5 per cent. glucose) for a week or two for the nausea and vomiting of pregnancy, and goes on in a perfectly normal manner.

On the other hand, pregnancy does have a remarkably stimulating effect on many patients, and it is no infrequent thing to see a slightly built girl develop wonderfully with excel-

lent color and increase in her hemoglobin, increase in the size of her bones, as the result of pregnancy. The anterior pituitary, the adrenal cortex, and the thyroid are stimulated and act well. The matter is rather different in the postpartum stage. Some patients are characterized by evidences of glandular exhaustion, which is intensified in many patients by nursing, especially if prolonged. If prolonged, we have the condition known as lactation atrophy, in which menstruation has been inhibited during nursing by the action of the mammary extract. The act of nursing separates from the mammary glands certain substances which have an inhibiting and restraining influence on the ovaries, posterior pituitary, and uterus, leading to an atrophy or subinvolution. There may be a marked constitutional effect associated with this prolonged nursing, the evidence then being a marked deficiency of the stimulating secretory glands of the body, such as the ovary, thyroid, and especially the pituitary and the adrenals. It is not very difficult to restore menstruation due to lactation atrophy by the administration of ovarian extract plus thyroid extract. The element of exhaustion or asthenia is of importance, and in these cases we add suprarenal extract and pituitary extract, *whole gland*, for its general tonic effect. Some patients have many children, even in rapid succession, and are perfectly well and in nowise exhausted. Some women after one baby suffer exhaustion which, while it may improve, is more or less permanent. In other words, certain individuals are potential asthenics, and pregnancy and labor add the final blow, and they practically never recover from the effect of the drain which has been put on their protective glands by the invasion of the system by the placental secretion.

As a general proposition, it may be stated that a nursing mother who menstruates has an endocrine system less readily held in check than has the mother who is characterized by amenorrhea during lactation. From this physiologic fact we deduce that mammary extract ought to be of value in many forms of menorrhagia due to ovarian pituitary or uterine causes, and experience proves this deduction to be correct.

What is labor?

Labor is a crisis, and in that crisis certain glands—particularly the ovary, thyroid, and pituitary, which normally are concerned in menstruation and which have been inhibited by the trophoblast and placental secretions—again come into action, with the result that on the two hundred and eightieth day you have a menstruation which did not take place during the duration of pregnancy. Something occurs which brings ovary, thyroid, and pituitary back into their old function. The inhibiting element is gone. Now, menstruation is a miniature labor. The processes of dilatation of the cervix, expulsion of non fecundated ovum, loss of blood, are miniature reproductions of the various processes which occur in labor. Therefore we have pituitrin acting when the patient is in labor, whereas it does not act until the crisis occurs. If given when the patient is in labor, it increases the pains, adds to their force, and its action is generally all that can be desired. Some inhibitory element has disappeared or some sensitizing element has come into being. If you give pituitrin before the patient is in labor, or before her expected time, it may bring on labor pains, but in the large majority of instances it has no further effect.

Why does it act when given by hypodermic during labor or at the time the patient is supposed to go in labor, and not at an earlier date? Because labor is a crisis, and you will find that pituitrin, given in proper doses and at proper intervals during labor at a time when forceps seem to be indicated, almost entirely displaces the need of forceps.

Now, we come to the question of repeated, habitual miscarriage. We have laid much stress on the meaning of the Wassermann test in this type of patient, but there are any number of cases of habitual miscarriage in which the Wassermann test is negative. Where formerly I used thyroid extract plus arsenic, plus bichlorid of mercury, plus stypticin for months to carry a patient to full term, I now find the results even better by relying almost entirely on endocrine treatment—giving the patient the trophic support which she needs and increasing the resistance to those factors which tend to produce miscarriage.

What is the cause of repeated miscarriages?

The ovary, thyroid, and pituitary are trying each month to produce menstruation, the placenta is trying for two hundred and eighty days to inhibit it. If the ovary, thyroid, and pituitary, especially the pituitary, are not too energetic, the placental element nullifies the menstrual tendency, holds it up for two hundred and eighty days, until labor ensues at the expected time. If the placental element cannot inhibit this tendency to menstruation, you have a menstruation early in pregnancy, which is a miscarriage, at two months, three months, four months, etc., so the whole is a matter of when the crisis occurs. If it occurs at the expected time, you have a full-term pregnancy, if it occurs earlier, you have a miscarriage, or premature labor.

So we give to these patients the secretions which have a tonic influence and a trophic effect, we give no pituitary extract in any form, for we fear the too early evidence of the action of that gland. We give mainly ovarian extract, corpus luteum, and thyroid.

Now we know that in hyperthyroidism patients may be improved by the administration of pituitary extract. If the reverse be the case and you have hyperpituitarism, you may oppose the specific action on the pregnant uterus by the administration of thyroid, so that the administration of the latter extract in repeated miscarriages may be explained by this trophic action and by its opposing the excessive and too early sensitized introduction of the pituitary element.

The introduction of pituitrin into obstetrics has marked a momentous advance. Labor-pains and the actual force which the uterus is exerting in expelling the fetus are two different things. A patient may have little pain, and progress may be normal, she may have no pains at all, worthy of the name—uterine inertia, she may have very severe pains, with no progress. The use of pituitrin in 3-minim doses, with the pelvic measurements normal and the head engaged firmly in and through the brim, and well molded, will deliver the vast majority of babies without the use of forceps. Obstetrics has

been too little surgical in its trend, gynecology has been ~~more~~ surgical

We turn to cesarean section as an example. In those cases where the test of labor aided by pituitrin shows no ~~engagement~~ ~~in~~ no engagement of the head in and through the brim ~~we~~ ~~do~~ rather than apply the forceps on such a head. The ~~indication~~ for cesarean section is even more clear when the ~~head does not~~ engage at all, but bobs above the brim. In placing ~~down~~ ~~in~~ a viable child, cesarean section has every advantage in that it saves the baby and is far less dangerous for the mother. When dealing with a clean case handled under sterile ~~protection~~ ~~protection~~ if examined, cesarean section, in my opinion, is preferable to the use of high forceps. So, with the ~~introduction~~ ~~of~~ ~~the~~ surgical element into obstetrics and with the ~~introduction~~ ~~of~~ ~~the~~ use of pituitrin, we find forceps less and less ~~used~~.

Why is gynecology getting to be less ~~surgical~~?

lacerated cervix? And this criticism holds good for many of the minor gynecologic conditions on which operations have been done so recklessly in the past. We must remember that lacerated feelings are much more responsible for nervousness than a lacerated cervix or perineum can be.

We now explain these so-called cases of hysteria and neurasthenia, and the innumerable and various nervous phenomena with which women suffer, on the very practical theory of glandular upset, and among these upsets hyperthyroidism, so frequent in women, proves to be the most frequent. The relation of ovarian secretion—altered by changes in the circulation, by slight degrees of inflammation, by the development of the so-called cystic ovary—to upset in the glandular action is only too clear. Changed ovarian secretion may be the cause of glandular upset, and altered ovarian secretion (glandular upset) is often the result of pituitary or thyroid abnormalities, even to the degree of anatomic cystic changes in the ovaries themselves.

Then we come to the menopause state, with the types varying from the depressed to the excitable, and to the combination of these two states. All of these are now to be explained by interglandular changes and alterations which occur in every woman at the climacterium. If these changes develop harmoniously, the patient has a placid menopause. If the new relationship established between the glands by the diminishing function of the ovary and the coincident diminishing function of the thyroid, pituitary, and other glands is not established easily and harmoniously, we have hyperthyroidism, hypothyroidism, hyperpituitarism, hypopituitarism, and innumerable combinations involving these glands as well as the suprarenal, pancreatic, and other structures. That many cases of glycosuria are the result of the interglandular changes, and that an excess of pituitary action likewise is responsible for many cases of glycosuria and fibroid uterus seems more than probable.

Now I will devote the rest of this talk to the subject of sterility.

Figure 174 shows the uterus in normal position with a moderate degree of anteflexion. It shows the posterior fornix, which

is supposed to represent a pouch or culdesac into which the seminal fluid is deposited, so that the cervix by dipping into it gives easy opportunity for the ingress of the spermatozoa. Some have considered one of the causes of sterility to be an anomaly whereby there is an angle instead of a pouch at the posterior fornix. On the theory that the seminal fluid runs out readily they have considered this anomaly to be one of the causes of sterility, and have treated these cases by successive packings of the vagina with gauze, and in that way believe they have developed a culdesac. So far as my observations have gone I have not found this to be a matter of any great importance.

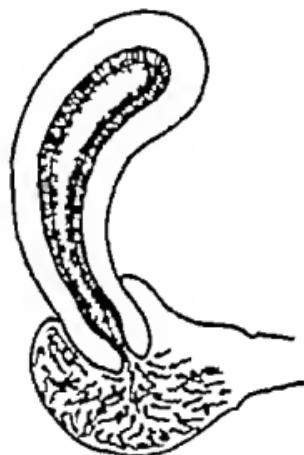


Fig. 174.—The spermatozoa are deposited in the posterior fornix.

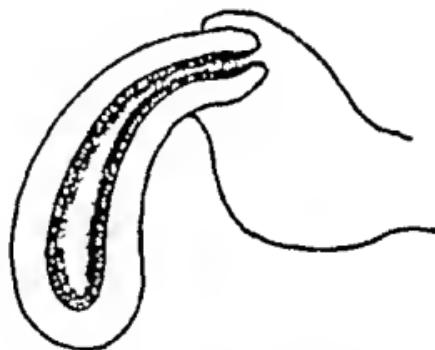


Fig. 175.—With retroflexion the cervix is not in contact with the reservoir formed by the posterior fornix.

Then comes the question of retroflexion (Fig. 175). If you are dealing with a retroflexion, as has been depicted above, with the cervix pointing horizontally or upward, you have naturally less opportunity for the spermatozoa to be in continuous contact with the cervix, and many men have considered and do consider retroflexion a frequent cause of sterility. It is, of course, the correct idea to replace every retroflexed uterus with the aid of a pessary. The trouble is that most congenital retroflexions cannot be restored to the normal position by this means. The cervix is high up and nearer the symphysis than is normal because the anterior wall of the vagina and the uterovesical ligaments are congenitally

short. To replace the retroflexed uterus it is essential that the cervix should be pushed high up and far back in the direction of the hollow of the sacrum. If then the fundus fall forward, as it naturally will if movable and not held by adhesions, the proper pessary by holding the cervix in this natural position mechanically corrects the retroflexion.

The next chart brings us to the question of inflammation (Fig 176). You may have inflammation of the cervix with or without a very thick plug of yellowish, greenish, mucoid secretion, you may have the inflammation affecting the lining of the uterus, the cornual or interstitial area of the tubes, the entire lumen of the tube,

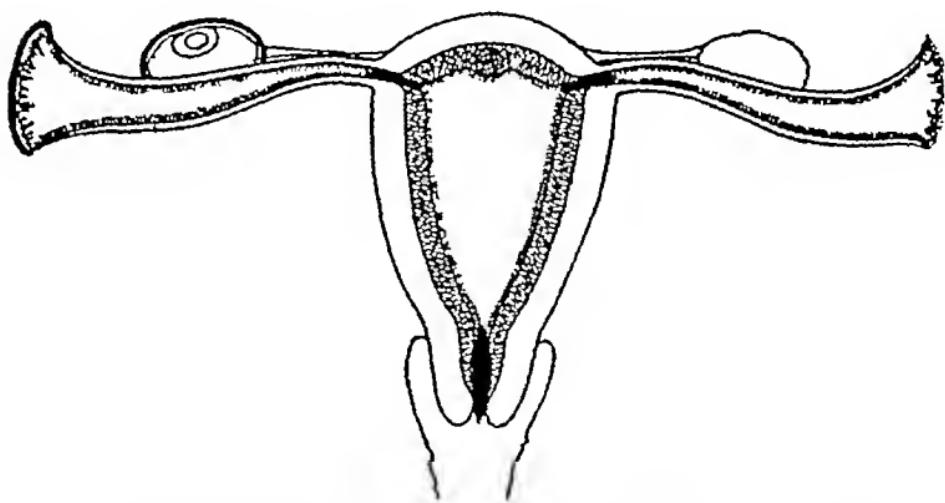


Fig 176.—The dark lines show the location of inflammation—cervix, uterus, tubal cornua, adhesions about the tubal openings, about the ovaries

with or without adhesions about the outer ends, with or without closure of the outer abdominal ends, you may have inflammation and adhesions about the ovary. If you have a yellow, greenish, thick, tenacious mucus pouring out of the cervix like a veritable Niagara Falls, you will find it impossible to readily remove it or to free it from the cervical canal. If you examine it with a microscope, you will get a pure smear of pus. To expect any spermatozoon to pass that obstacle, leaving aside the inflammation, is to ask too much. If you dilate a cervix of this kind, or any inflamed cervix, you are stirring up an active or a latent inflammation. If you curet a uterus whose lining is involved

by inflammation, you are doing the same or worse, for every gonorrhreal cervicitis or endometritis lights up after a curetage. The bacteria thrive on the blood which is poured out, for human blood and human serum are the best culture media for gonococci. If on bimanual examination you conclude there is no salpingitis—and such a diagnosis must be made cautiously—how can you exclude the existence of a salpingitis in the cornual area? One certainly cannot deny the possibility that it is present in that part of the tube. If by any chance, there is no evidence of an endocervicitis, and no marked signs of an endometritis, you cannot definitely deny the possibility of these conditions having improved without treatment in the course of time, and that they have left slight evidence behind. The milder forms of salpingitis may exist without any cervical signs.

Hence, if you curet any of these cases, you drive the inflammation from the uterus into the tube, or from the cornual area of the tube out through its whole length into the peritoneal area, adhesions form, and the very thing you should avoid has happened. You should never curet a uterus for sterility or dysmenorrhea, in fact, you should never curet at all until by repeated examinations you exclude an inflammation, and you should not do it any way when a patient's menstruation is normal or below normal. One thing must be impressed with the greatest emphasis and that is, that you cannot cure an inflammation of the cervix or uterus by curetage. The lining of the uterus is not a membrane to be toyed with on the principle that it is readily restored. Even if no inflammation is present, a curetage may so affect the ovary that the patient suffers subsequently from varying degrees of amenorrhea. As between the endometrium and the ovary it is a case of "you tickle me and I tickle you." The ovary stimulates the endometrium, the endometrium grows thicker, the cells grow larger in anticipation of the next menstrual decidua, and the secretion thus produced in turn stimulates the ovary. If you remove too much endometrium, the ovary is injured and may atrophy. Except in cases of polyps or polypoid endometrium, in cases of fibrosis uteri where pregnancy is no longer a consideration—and here only after the exclusion of in

flammation—I have not cured to any degree for years, and even in miscarriage the curet is scarcely ever used I hope that some day there will be a law that before we curet a patient we must get a license or permission from some central authority to use it in a particular case and then only after giving genuine reasons therefor

Now, leaving the element of inflammation entirely out of the question, how about cervical stenosis, angulation at the internal os, etc., as the cause of sterility? The first questions always to be answered are Are normal spermatozoa present? Is a ripe ovum thrown out? How do we know whether an ovum is thrown out at every menstruation, or even at irregular intervals? Between menstruations a ripening Graafian follicle grows so that it reaches the surface of the ovary, separated from the peritoneal cavity by only a thin membrane This membrane (the outer covering of the ovary) is dissolved by an enzyme action of the liquor folliculi which erodes, allowing the fluid to be expelled, carrying with it the ovum But suppose a follicle lacks that enzyme, and that it does not burst, does not liberate the ovum? It remains in the ovary and is an atresic follicle If this process is repeated, one or both ovaries are filled with these little cysts, and it is often difficult or impossible for such ovaries to free any future ripening follicles as they attempt to approach the surface

Why do certain ovaries lack this enzyme action of the follicle? Because something is wrong with the thyroid, adrenals, or pituitary which support the ovary in its activity In addition, you have the inflammations whose invasion of the ovary leads to the type of small cystic degeneration Hence we come to the conclusion that even a normal menstruation may not mean the throwing out of an ovum

Now, if a woman menstruates only every two or three months, or at longer intervals, does that mean that she gives out no ova? No I have seen many such patients become pregnant without any regularity in their menstruation at all How about the woman who becomes pregnant while nursing a baby and who during this nursing does not menstruate? A nursing mother with amenorrhea is not supposed to be ovulating because the

action of the mammary secretion inhibits the function of the ovaries, and probably at the same time exerts an inhibitory action on the pituitary lobe, yet such women do ovulate, and we have thereby a proof that ovulation may take place without menstruation, so that varying degrees of amenorrhea, or even the more decided degrees of amenorrhea, are not necessarily a bar to fecundation.

Now, suppose that we are certain that an egg is thrown out from an ovary every month, and suppose we are sure from examination that normal spermatozoa are present, what gives us the right to locate the obstacle to fecundation in the cervix? May

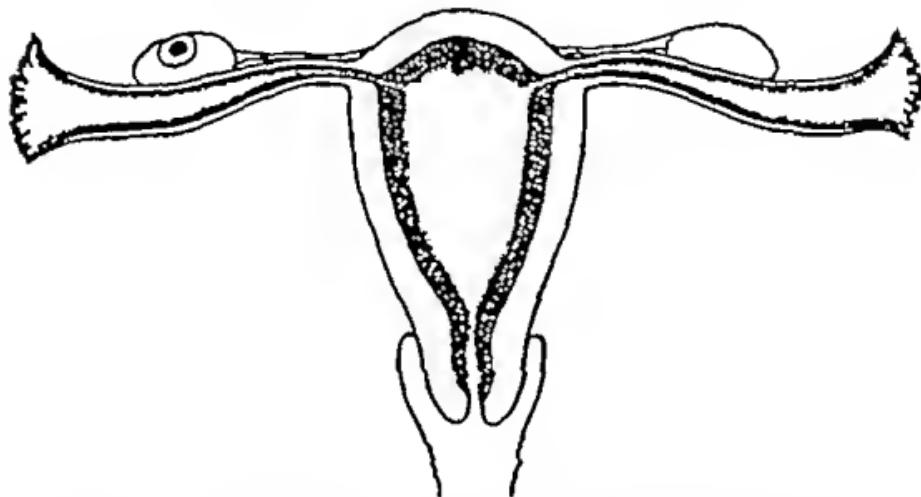


Fig. 177.—The ovum given out of its follicle moves through the tube, carried along by the cilia, and finally nesting in the lining of the uterus.

it not be the tube which is at fault because the cilia do not act? May it not be a failure of embedding and nesting of the ovum? Let us for a moment consider the earliest steps of pregnancy. Spermatozoa are deposited in the posterior fornix (Fig. 177). They pass up through the cervix by virtue of their own activity against the current of the ciliated epithelium in the endometrium. As you know, the ciliated epithelium in the endometrium waves downward toward the cervix. It is the nature of an energetic individual to surmount obstacles and not to always follow the current of ease. Men who make the biggest successes are those who do it in spite of opposition, and we may take these tiny

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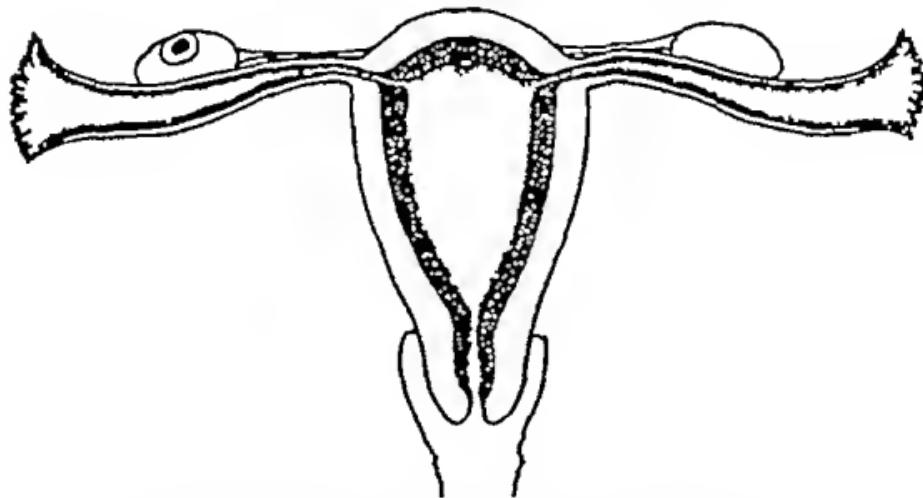


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spermatozoa as an illustration. Why do they pass upward through the cervix into the uterus? It is because the current is against them, and they meet the same opposition in the tubes, for there the cilia wave from the abdominal end toward the uterus. If you cut the uterus at the interstitial area and see the caliber of the tube at this point you will see how extremely narrow is the lumen. If spermatozoa pass out through this canal and if fecundated ova are moved through it by the cilia into the uterus, why cannot a cervix that readily admits a sound offer a ready path for the upward moving spermatozoa? If a cervix is filled with adenoid overgrowths of the mucosa, if the internal os is lined by a lymphoid ring of this overgrown tissue, it is possible that the obstacle to the spermatozoa may be absolute, but in the non-inflammatory cases I believe we have tremendously exaggerated the rôle of cervical obstruction, and I have laid relatively little stress on the cervix as a cause for sterility.

Now, what happens when an ovum leaves the Graafian follicle? It is discharged into the peritoneal plasma and is drawn into one tube or the other by the current created by the cilia of the outer end of the tube waving toward the uterus. Through the tube it is pushed along on the tops of these cilia, and now we can readily understand why, if you have a tube on one side of the uterus with the ovary removed and a normal ovary on the other side with the tube removed, an ovum can be drawn from one side of the uterus over into the outer end of the tube on the other side. Inside of the tube is a microscopic ovum, inside of the tube are microscopic spermatozoa. It is interesting simply to wonder what attracts the spermatozoon to the ovum, so that the head of the spermatozoon may enter the ovum and fecundate it. When the egg is fecundated it enters the uterus, the outer cells of this growing ovum forming a cell layer called the trophoblast, which digests the surface of the decidua at the point where it is to settle. It burrows itself in this overgrown thickened mucosa, is surrounded by lymphoid exudate, and covered by a tiny scar. When this occurs, the trophoblast cells throw off a secretion which is absorbed into the circulation, and this secretion continues to be thrown into the mother's blood during the

whole nine months of pregnancy. The chorionic villi develop especially at the point where the ovum rests, and here opens the future placenta. Hence if an ovum settles at the lower segment of the uterus we have the varying forms of placenta praevia. The secretion given off by the outer covering of the ovum nullifies the action of the ovary, thyroid, and especially the pituitary, which normally lead to menstruation. If this fecundated ovum cannot nullify this menstrual tendency, blood is poured out from the capillaries and the ovum is thrown out. In other words, many a fecundated ovum, by its inability to resist the menstrual stimulus, fails to remain in the uterus more than a few days. We have many patients who occasionally or repeatedly are a week or ten days over the period, and then menstruate, who undoubtedly represent this process. I have often told such patients not to worry, that some day by the aid of the internal secretions administered therapeutically we would so help the trophic processes concerned in embedding and nesting that normal pregnancy would take place, and, sure enough, it often does.

active, they will not carry an ovum into the tube. Why not also take it for granted that some of the follicles of an ovary would break if you gave them sufficient trophic support. If we conclude that sterility is due to affections of the ovary, thyroid, and pituitary gland, and that ovary supported by thyroid and pituitary is concerned in the care of the genital tract and that they act together in the production of menstruation, then we must stimulate the genital tract by extracts of these glands. Whether they aid the bursting of follicles which would otherwise be atresic, or whether they stimulate the ciliated epithelium of the tubes to normal activity, or whether they aid the processes of embedding in a trophic way, is of no great moment in any successful case. So long as in many favorable cases, where two or three of these gland extracts are given, a favorable result is obtained, what difference does it make to us which function was aided by the therapy—whether the ovary, the cilia of the tubes, or the element of embedding?

Chart No. II discloses some of the knowledge we have concerning the ovary and other secretory glands, based on animal experiments made many years ago. I was fortunate enough to be acquainted with Knauer at the time he made those first studies on animals, with particular reference to the ovary. Ovaries were taken out of the young, with the result that the genitalia failed to develop. Ovaries were taken from mature animals, and the genitalia underwent atrophy. On the other hand, ovaries removed from their normal connections were transplanted into the mesenteric region or between the fascia of the abdominal wall, and whenever these were properly nourished and produced follicles the genitalia developed if the experiments were made in the newborn, and the genitalia were trophically sustained if these operations were made in mature animals. This proved that the ovaries exert their action by virtue of the secretion sent into the human economy.

You know the old idea concerning menstruation was that the ripening Graafian follicle distended the ovary, and the impulses from the ovary were transmitted to the spinal cord and thence to the uterus, causing the premenstrual and menstrual phenomena.

This would make menstruation dependent on the rhythmic and regular liberation of an ovum at or about the same time, and I think we have shown in our discussion already that ovulation, while it usually precedes menstruation, does not always do so, and that menstruation is dependent upon ovarian secretion liberated continually and continuously.

It is the result of these experiments and the results obtained by cauterizing the corpora lutea in the early months of pregnancy in animals (with the resulting expulsion of the uterine contents) that attracted attention to the corpus luteum as an important trophic element in connection with the genitalia and pregnancy.

We now come to the climacterium and its usually associated diminishing or disappearing menstruation, with a certain proportion of cases characterized by excessive menstruation. Normal menstruation represents harmonious team work on the part of the ovary, thyroid, and pituitary. As we have stated before, children with thymus affections, certain types of thyroid disease, definitely recognized pituitary anomalies, fail to develop their bones, or bodies, or their minds. Affections of this sort are not always of the major type with well marked characteristics. There are varied and minor forms where only one or other of these phases is apparent, development is not parallel as regards all the functions, the affection may not have been sufficient to interfere markedly with the general appearance or health of the individual, and yet the genital development may be relatively negligible and may represent any of the varying degrees of hypoplasia. Then we have the woman or girl who has developed normally, who has menstruated regularly, and then, on the development of a thyroid, pituitary, or other glandular anomaly, has suffered a deterioration in the region of the sexual organs. Then we have the case of pineal, adrenal, and pituitary gland tumors characterized during life by marked precocious sex development, and tumors of the above mentioned glands have been found at autopsy to have so altered the secretory activity of these important structures as to leave us in no doubt that these glands were responsible for the excessive development in the

sexual sphere. That leads me to believe that if children were fed with certain gland extracts at certain periods, with a definite purpose in mind, we could make of them physically, mentally, and otherwise far healthier, stronger individuals than when left to the mercy of their own internal secretory apparatus—which with every passing year we recognize to be in many instances inadequate to the needs of the body. The diseases of childhood,

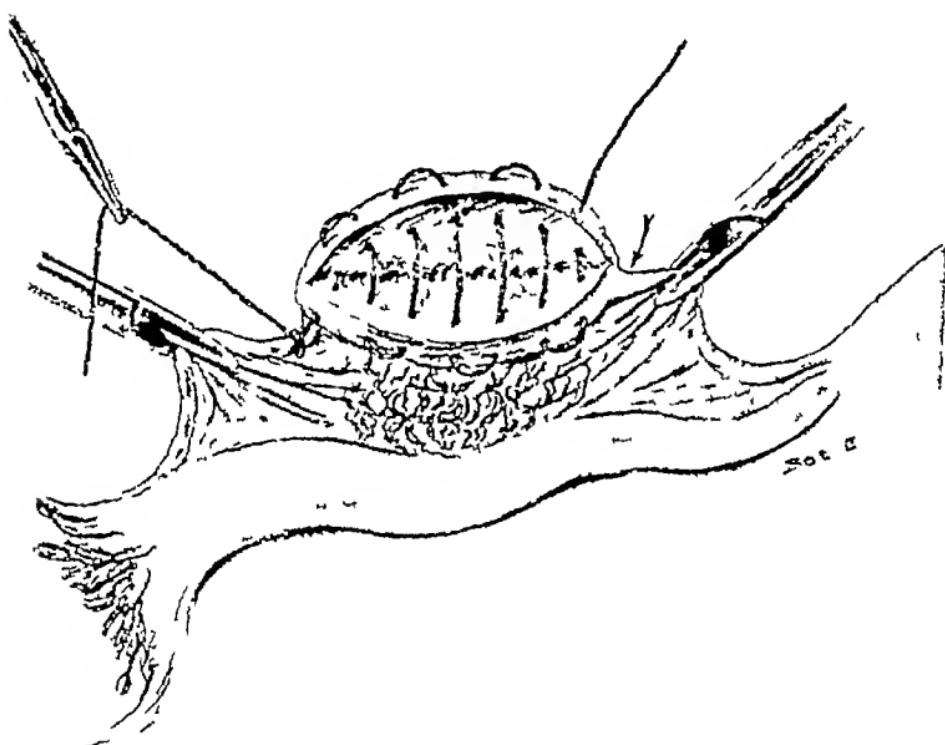


Fig. 178.—A wedge shaped area is taken out of the convex surface of the ovary, removing all the cysts and corpus luteum rests. The raw surfaces are brought together by continuous suture

such as whooping-cough, measles, scarlatina, diphtheria, mumps, etc., have in many instances an injurious effect on one or more glands of the body, and from our standpoint injury to the ovary and the uterus, and those glands which are associated with the trophic control over the genitalia, deserve the greatest consideration. Many cases of uterine and ovarian hypoplasia are to be referred to the injurious action of the diseases of childhood. If the toxic effect of these infections is centered on those elements

of the glands which preside over the mental and psychic states, we have an explanation of many of the mental and nervous affections of children for which no apparent cause has heretofore been brought under consideration

We refer again to the harmful effects of the present influenza epidemic on the uterine mucosa, with its influence on menstruation. It is natural if that same injury takes place when the growing ovum is in the uterus that a tendency to miscarriage or premature labor should occur. I have had several cases of

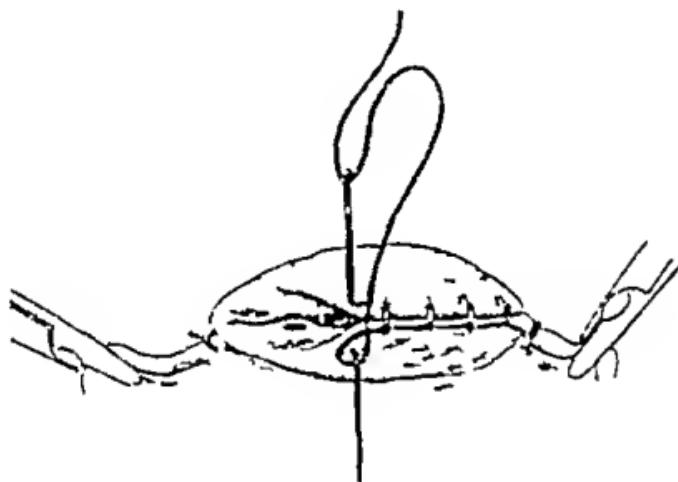


Fig 179.—An interlocking suture covers the raw edges after the preliminary suture shown in Fig 178.

pregnant women who after slight attacks of influenza began to spot and stain. They were put to bed, given a dose or two of morphin, and immediately put on ovary and thyroid extracts, with excellent results

Figures 178 and 179 illustrate my operation for exsection of the cystic area in the so-called cystic ovary. Let me mention a patient who was sterile, married six years. Normal spermatozoa were present in the seminal fluid of the husband. Inflammation, judged by the severest tests, bimanual and microscopic, was excluded, no tubal inflammation was found. The patient was

put on endocrines, with no effect upon the sterility. My conclusion was that the endocrines, by their effect on the tubes and endometrium, have excluded inactivity of the cilia and failure of proper embedding as the causes of sterility, therefore ovulation does not take place in this patient. I opened the abdomen and found both ovaries cystic. Unless you find an actual corpus luteum from a recent ovulation present, you cannot tell from observation of the ovaries whether ovulation takes place or not. I removed a wedge-shaped piece from each of these ovaries, leaving behind only normal tissue free of cysts. The patient became pregnant within a few months. Therefore, just as I take out the appendix in every laparotomy, I excise this wedge-shaped piece from the cortex in every cystic ovary where laparotomy is performed, no matter for what purpose.

Figure 178 illustrates the method of sewing the ovary after excision. The No 1 catgut suture begins at one end with a knot, and then passes through and through the ovary, uniting the raw surfaces together by a Connell suture until the other end of the excised area is reached. Then a knot is tied on the free edge of the mesovarium. The continuous suture then goes back over the raw edges (interlocking in character) until the first knot is reached, when the two ends are tied (Fig 179). It is essential to be sure that hemostasis is complete, and an occasional interrupted suture is needed to obtain this result. I believe that the time is coming when this operation will be done very frequently, with sterility as the primary reason for same.

If we could assure our sterile patients with certainty that this operation would be successful we would find a ready response, but, however certain you may be, by excluding other causes and by the failure of endocrine therapy, that this ovarian condition must be the point at fault, it is too great a responsibility to guarantee success after this operative procedure. Several of my patients, however, have become pregnant after this operation, and even if to the carping critic I have not proved that the operation was the cause of the pregnancy, it certainly does prove that the patients may become pregnant after resecting a wedge from each ovary, and even after removing one ovary and resecting

a wedge from the other. In one patient I removed a dermoid cyst of one side and half of the ovary from the other, and she has since been delivered of two babies.

We must remember that when these cystic ovaries contain corpus luteum cysts, or corpus luteum nests, ovulation may be inhibited, that is the purpose of the true corpus luteum which develops after pregnancy takes place. This true corpus luteum body, supposed to aid and further the connection of the ovum with the uterine lining, is supposed to inhibit ovulation during pregnancy. So if corpus luteum bodies are retained in one or both ovaries, even though menstruation continues, I believe in many cases ovulation does not. Therefore, in removing from these ovaries not only the atresic follicle cysts but also the corpus luteum cysts, or corpora lutea, I believe we are allowing the processes of ovulation to be again restored.

The following chart shows some of the facts proving the relation of gland states and the infectious diseases of childhood and adult life to the development and conservation of the genitalia.

CHART No. II

- 1 Removal of ovary { Failure of genital development.
Genital atrophy
2. Transplantation of ovaries { Genital development.
Preservation of genitalia.
(Internal secretion)
- 3 Operative removal of ovaries Menstruation not restored by endocrines.
Diminished menstruation—menopause.
4. Climacterium { Increased menstruation = ovarian and pituitary over activity
5. Physiognomy of menstruation { Ovary supported by thyroid and pituitary
(Crisis)
6. Action of infectious diseases of childhood Retard development.
- 7 Action of thymus, thyroid, and pituitary diseases } (a) Direct, (b) through
on the development of the genitalia } the ovaries.
- 8 Action of thyroid and pituitary diseases on the preservation of the genitalia.
- 9 Pineal adrenal and pituitary precocious sex development
- 10 Action of influenza on menstruation and pregnancy

What are the facts of practical experience which tend to prove the value of endocrine therapy? It is over eighteen years

ago since I began to use ovarian extract and thyroid extract. Since then various other gland secretions have been put into our hands, and the experience gained by the successive use of the various extracts singly and combined has really meant the entire remaking of the therapy in gynecology. It is certainly one of the greatest pleasures in a medical man's life to have a woman menstruate regularly when her complaint has been a menstruation every five or six weeks, and then only for a day or so. Other women menstruate every three months, some, once a year. While some of these cases are not curable, if the process of atrophy has gone on too far or if the hypoplasia is too marked, yet in a large majority of instances the administration of ovarian plus thyroid plus posterior pituitary extracts, etc., with patience brings about the desired result.

Following is a little chart showing some of the facts which favor endocrine therapy

FACTS FAVORING ENDOCRINE THERAPY

OVARY, THYROID, PITUITARY ANT., PITUITARY POST., THYMUS, MAMMARY,
SUPRARENAL, ETC

- 1 Action in varying degrees of relative amenorrhea
- 2 Action in varying degrees of actual amenorrhea (no signs of endocrine abnormality)
- 3 Action in varying degrees of actual amenorrhea Signs of endocrine abnormality—pituitary, thymus, etc.
- 4 Action in varying degrees of menorrhagia
- 5 Action in threatened miscarriage.
- 6 Action after repeated abortions

Aids attachment
 Trophic.
- 7 Action in lactation atrophy
- 8 Action in aiding fecundation—after one baby, with varying periods of delay
- 9 Action during menopause of climacterium
- 10 Action in sterility

In speaking of sterility, we should make clear to ourselves what time must elapse in the marriage state before the patient can be put in the category of sterility

I formerly took it for granted that if a patient was married a year and a half and was not pregnant, serious medical consid-

eration should be given to her condition, but today I have concluded that if a woman does not conceive within six months after marriage when no precautions are taken, or within six months after precautions are no longer taken, that sterility really exists. So, in taking the history, never fail to inquire not only how long the patient has been married, but for how long a period precautions were taken.

In order to arrive at the proper conclusions I took the cards of 80 of my own primigravidae, and found that an astonishingly large number had been married from ten months up to four years, and several five and ten years, before fecundation took place. I then inquired directly or by letter as to how long a period precautions were taken, and subtracting this period I found that practically 95 per cent. became pregnant within three to six months, the very largest number, between one and three months.

One patient was married ten years and occasionally accompanied a pregnant patient of mine to the office. She had taken precautions. A former friend of hers had had such a difficult labor that she was too frightened to allow herself to conceive. When she saw her friend, this patient of mine, delivered with relatively little pain, she concluded that labor was not such a terrible condition, precautions were avoided, and she conceived at once.

Two patients who had taken no precautions conceived—one after eleven years of married life, the other after ten years. The former had visited many physicians, had been advised to have a dilatation and curettage done, had been told she had a floating kidney and a retroverted uterus, in fact, she had never consulted a physician without operation in one form or another being urged. Fortunately for her, she never followed any of the suggestions that were made, and as her health was poor she was given a rest cure, in the course of which she gained 30 pounds, and a few months later she conceived. The explanation is to be found in one of two possibilities. Either she had a form of salpingitis affecting the interstitial area of the tube which in the course of time healed, or some interglandular upset was righted in the course of time, and some trophic change occurred in the

uterus or the tubes, or in the ovary, that permitted ovulation, the carrying of the ovum into the uterus, and embedding, to take place

The other patient was operated upon eight years ago—after being married two years—for double-sided pyosalpinx. One tube and ovary were removed, the tube of the other side was cleansed, and with the corresponding ovary was left in place. At the time I am now speaking she is three months pregnant. In other words, it took nature eight years to heal up the remaining tube sufficiently to restore its patency and restore the activity of the cilia.

In another portion of the chart you will see 5 patients, 4 of whom had half of each cystic ovary resected, while the fifth had a dermoid of the ovary and the tube of one side removed, and half of the cystic ovary of the other side. All have conceived. The operations were done for sterility.

In the table shown on page 951 are 50 consecutive cases of sterility. No matter what other complaints the patients had, I have taken no records except of those who when asked by me as the history was nearly completed, What are the two things of which you are complaining? gave as the first answer "Why do I not become pregnant?" In other words, these patients came for an answer to this question.

In the first column you will see the patients in whom I found inflammatory conditions sufficiently marked to lay the blame on this state, with little reference indeed as to the question of spermatozoa. The diagnosis is written after each name. The names surrounded by a circle have been laparotomized by me, and the diagnosis in every instance verified—inflammation, with closure of the tubes.

Underneath you will notice two patients—one married ten years and the other nine months—whose conditions warranted operation. The first had a double tubo-ovarian cyst filling out the entire pelvis, the other, married nine months, whose menstruation lasted only one day, had a large fibroma of one ovary and a small one of the other.

The next column shows the cases in which the male element

The table shows four columns, the first being a list of cases in which inflammation of the tubes and ovaries were the cause of the sterility, the diagnosis was verified by operation in the cases which have a circle about the name. The two cases at the bottom of the first column were cases of tumor of the ovary both were operated on and found. The third column shows cases in which the spermatozoa were no longer found. The fourth column shows the cases where menstruation was markedly diminished and where in spite of endocrine therapy, no

The last column shows successful cases, the first five becoming pregnant after a resection operation on the ovaries the others becoming pregnant after endocrine therapy only. The figures before and after the initials in each column show how long each patient was married the figures before the initials meaning years after the initials, meaning months.

was either lacking or so deficient that the responsibility for the sterility does not rest on the female

In this column you will see a vertical line after some of the names, which means that they were either cured or some cervical operation was done on them. It is needless to say anything further. One of these patients was cured twice. No criticism is too severe to be vented on a physician who curets or dilates, or does a cervical plastic for sterility without first finding out whether or not spermatozoa are present.

The next column shows those patients in whom endocrine therapy has so far failed to result in pregnancy. It is needless to say that examination has proved spermatozoa to be present and active. With the exception of 2 patients, one of whom was cured several years ago, none menstruates normally either in amount or with any degree of regularity. One menstruates every seven months, another, every six weeks for one day, another, every four to seven months, etc. Three of these patients are of the type of *dystrophia adiposogenitalis*. Under endocrine therapy menstruation has improved in practically every one, but as yet only one has conceived since this table was made out.

Now, as to the question of operation on these patients—whether they have cystic ovaries or not, or whether they ovulate or not—I have come to the conclusion, as a result of operation on several of this type of patient, that resection of part of each cystic ovary promises a favorable result only in those patients whose menstruation is regular, normal in amount, or excessive.

The next column shows the patients who have become pregnant 5 after operation on the ovaries, and 12 as the result of endocrine treatment.

Patient H was married eight months, had taken no precautions, and was in tears because she had not conceived. Her menstruation was excessive, lasting six days, with clots. The uterus was not large, but because of the excessive menstruation I gave her thymus extract, which she began to take two weeks before the next expected menstruation, which, however, she passed, and nine months later was delivered of a baby.

The next patient was married nine months. Her menstruation was only slight, lasting a day or two. She was given ovarian and thyroid extract, became pregnant, and was delivered of a healthy baby.

The next patient was married nine months. Menstruation was normal. She was put on ovarian and thyroid extract two weeks before the next expected menstruation. Her husband was requested to send me a condom specimen. She passed her next menstrual period, and is now under my care awaiting delivery.

Mrs. S. H. D. was an interesting case. Here was a patient who menstruated every three months. As the result of endocrine therapy she menstruated normally for two periods, and then came to me after a lapse of three months saying that the medicine had failed to produce a regular menstruation and that she had relapsed into her old state of amenorrhea. Examination showed that she had a genuine cause for this last amenorrhea of three months, the cause being a growing ovum in the uterus. She completed her labor in a perfectly normal fashion, even though the duration of her pains was longer than usual.

It is with me quite a usual experience to note that those patients who become pregnant only after gland stimulation do need pituitrin and that the labor lasts longer than is usual. One would expect this as a matter of course, for all these patients are to be viewed as having a certain interglandular dysfunction. The most interesting was Mrs. R. O. S., who became pregnant after being married six years. When she first came to me she suffered from intractable menorrhagia which yielded only to a thorough curettage. For years afterward she menstruated at intervals of forty to eighty days, which yielded and improved under endocrine treatment. Because this patient finally became pregnant after having given up hope, and because we were dealing with a well-developed baby, and because on the basis of previous experience I feared the labor would be either very long and demand much pituitrin or would eventually need the application of forceps, I delivered her by cesarean section. The interesting point is that she suggested this operation of her own

accord, because she felt from what she had read of the subject that it was the safest, surest, and quickest means of insuring her a live baby

Of the cases which did not respond to treatment (col. 3), one menstruates every seven months, and was not at all faithful in taking the endocrines. Another menstruates every six months for one day, with the endocrines, her menstruation lasts two and a half days. Another patient, a pituitary dystrophy, menstruated every three or four months, but with endocrine menstruates regularly. Another is the most typical of pituitary dystrophies, who with the aid of endocrines given by mouth and hypodermically menstruates regularly for two and a half to three days. Another patient who formerly menstruated every four to seven months has continued the endocrines for a long period and her menstruation is almost normal.

So in these patients we are dealing with profound pluriglandular upsets, all characterized by diminished menstruation. It is for this reason that we do not consider them proper patients for abdominal operation on the ovaries, and my experience in this matter has shown me that we may expect a favorable operative result only in such patients as menstruate fairly regularly and in a fairly normal amount without endocrine stimulation.

Just a few words about the treatment of the male. If by substituting those endocrines which normally exist and normally exert a trophic and stimulating action on the ovaries, tubes, and endometrium we can thus aid the processes leading to fecundation and embedding, the same trophic and stimulating influences may be exerted on the structures which produce the spermatozoa, therefore, the same gland extracts should be used, with the substitution of testicular extract for ovarian extract, when the spermatozoa are not sufficiently active. In several cases I have treated the husband on this theory after examination of the spermatic fluid showed the spermatozoa to be not normal, and in the future I will give added attention to this phase of the question.

Of the gland extracts which are used, ovarian extract is given in doses of 7 to 10 grains of the whole gland. Ovarian

residue has been given in several cases by hypodermic—this being an extract of the ovary minus the follicular tissue I believe this to be a very important part of the ovary

Thyroid extract is given, as a rule, in doses of $\frac{1}{16}$ to $\frac{1}{8}$ grain three times a day Larger doses than this I rarely give

Suprarenal extract of the whole gland is given in doses of 2 grains. From a study of the histology of the adrenals, from our knowledge of its secretion, and from changes which occur in the cortex during pregnancy the cortex is the more important element from the standpoint of the gynecologist.

As to the pituitary extract, it is the posterior lobe which furnishes us with the element having a trophic and stimulating effect on the genitalia. The dose of this is $\frac{1}{2}$ to 2 grains three times a day Its effect in stimulating is pronounced It is the important part of the pituitary gland in the various types of amenorrhea. The anterior lobe, as already stated, has a marked influence on growth, and during pregnancy we frequently see evidences of its well known hypertrophy during this period in the growth in stature, in the acromegalic change of features, and in the tonic effect which it has From my observations, it has an effect on the female genitalia opposite to that obtained by the posterior pituitary lobe when administered, especially if mammary extract be added, it has a decided effect in reducing the size of the uterus Therefore I do not use it in any form of amenorrhea. If given for its tonic effect in any case, this fact should be borne in mind, and posterior pituitary or ovary, or thyroid should be added

Mammary extract is given in doses of 7 to 10 grains or more, three times a day, for menorrhagia, metrorrhagia, for fibrosis uteri, for myomata, or fibromata. As just stated, it acts particularly well when combined with anterior pituitary extract.

Thymus extract is given in doses of 5 to 10 grains three times a day It is of value in the various forms of menorrhagia and metrorrhagia due to overactivity of the ovaries, and possibly acts well when the posterior pituitary is overfunctioning

Placental extract, which is not yet on the market, but with which I have been experimenting for over a year and a half,

acts well in certain forms of dysmenorrhea and in certain forms of menorrhagia and metrorrhagia

With these preliminary statements in mind, one may understand the following drawing (Fig 180)

In the center are the ovaries and the genitalia. Beneath are the gland extracts, which lift up, support, or stimulate the ovaries and the genitalia. They are ovarian extract, thyroid extract, suprarenal extract, and extract of the posterior pituitary lobe

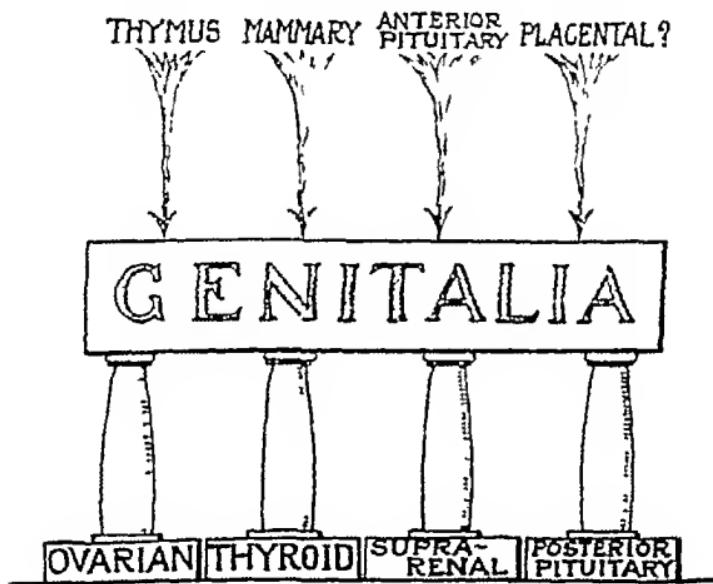


Fig 180

Above the ovaries and the genitalia are the gland extracts, which depress or inhibit the action of the ovaries and genitalia. These are thymus extract, mammary extract, anterior pituitary lobe extract, and probably placental extract

With this figure in mind, one can see the basis on which to build up a system for administering these substances when the functions of the ovary, tube, and uterus are under consideration. But this is not all that we gynecologists have to bear in mind in treating our patients. We have to take into consideration the symptoms of diverse types from which they suffer—their gastric disturbances, their premenstrual phenomena, their headaches, their nausea, their nervousness, their states of mental

depression and excitation, as well as their minor and major forms of psychosis. Every one knows the symptoms belonging to a typical case of Basedow's disease, and such cases may be recognized at a glance, but there are thousands of patients who have hyperthyroidism without exophthalmos or without goiter, or without persistent tachycardia, which nevertheless can be recognized as hyperthyroidism. There are still others who at one time or another under the stress of physical or mental irritations give evidences of annoyances which can be attributed only to temporary or transient attacks of hyperthyroidism. It is the recognition of these masked types which may or may not be associated with true gynecologic abnormalities that must engage the attention of all earnest medical men.

Undoubtedly, the greatest difficulty in the proper interpretation of interglandular upset depends upon the fact that so many of the cases are of minor degree—of a degree less than is typical of the well-exemplified cases.

If we have exophthalmic goiter on the one hand and myxedema on the other, gigantism or acromegaly on the one side, certain types of dwarfs or dystrophia adiposogenitalis on the other, if we have fibrosis uteri and fibromyomata on the one hand and dystrophia adiposo genitalis on the other, if we have tetanus and paralysis agitans on the one hand and myasthenia gravis on the other, if we have excessive sexual and physical development due to tumors of the pineal, the hypophysis, and the adrenals and testis on the one hand and cases of undeveloped genitalia and infantile uterus on the other, if we have acromegaly on the one hand and osteomalacia on the other, if we have excessive function and menstruation through oyster ovaries on the one hand and diminished function and relative amenorrhea through ovarian hypoplasia and degeneration adiposogenitalis on the other, if we have the extreme adrenal disease known as Addison's disease, why may we not expect minor degrees of involvement in the glands or in the glands responsible for the major cases, the resulting symptoms here often lacking the earmarks which define the standard types of disease not made mention?

If instability of gland function is transmitted, we have a new basis for predicating the various types of abnormality which may be inherited. Inherited instability of the thyroid may lead in the progeny to either myxedema or to exophthalmic goiter, or to variations between these two extremes. Inherited instability of the hypophysis may lead to small stature or to large growth, or to simply the psychic manifestations of dyspituitarism. *So that one member of a family may inherit from his parents or grandparents these somatic changes due to gland anomalies, or one may inherit the nervous tendencies or the instability of the nervous system, or psychoses resulting from anomalies of the internal gland secretions.*

We must distinguish between the somatic and the mental or psychic side of pathologic states due to the endocrine relation. I have seen in so many of my patients attacks of mental depression and blues, so many cases of excitement and states of exaltation of minor degree, so many cases where the states vary from slight exaltation to slight depression without apparent cause, cases after labor with depression of a mild melancholic type, that long ago I came to the conclusion that we must grant variations in intensity in mental diseases.

If we have the forms known as manic-depressive insanity, dementia praecox, melancholia, etc., why may we not have minor types of the same conditions confronting us in our gynecologic obstetric work? We know the excitability connected with the various grades of hyperthyroidism, we know the mental apathy associated with the various degrees of myxedema, we know the mental peculiarities and the changes in character in patients with hypophysis alterations. *All these variations, noted from time to time in my experience, have convinced me that mental diseases of extreme type may have the same relation to the milder forms and to the so-called neuroses and psychoses, and to the so-called neurasthenia and hysteria, that the major forms of exophthalmic goiter and myxedema, gigantism and dwarfism, etc., bear to minor variations noted every day.*

CONTRIBUTION BY DR. WALTER TIMME

NEUROLOGICAL INSTITUTE

A NEW PLURIGLANDULAR COMPENSATORY SYNDROME

INTRODUCTION

FROM observation and clinical examination of many patients throughout the past six years at the Neurological Institute of New York City I have come to the conclusion that the so-called types of endocrinopathies, such as status thymicolymphaticus, giantism, infantilism, acromegaly, and a host of unnamed others, are not static states, as one would be led to suppose from descriptions in the literature, but are simply cross-sections taken at intervals in a dynamic, progressive, and wide-spread disturbance of the internal glandular system. We had been struck at the Neurological Institute by the constant repetition of patients showing similar symptoms and similar physical signs which heretofore had been generally accredited to the asthenias accompanying neurasthenic states. The patients were usually of the late adolescent period, in the early twenties, and, to neglect for the present the detailed symptoms which we will go into later on, the outstanding complaints, overshadowing all others, were headache and muscular fatigability. Accompanying these two suggestive conditions there was an additional statement that they had been growing very rapidly for the past few years. We went deeply into their antecedent history and their family history and were enabled thereby to find many points of resemblance in these various individuals. One case, observed for six years, went through various stages to recovery at the age of thirty two years. From his early history we recognized symptoms that are presented by cases in the beginning stages of this syndrome, notably the headaches, fatigability, and the skeletal growth. Cross-sec-

tions at various stages of his further progress also resembled clinical pictures in patients that had formerly been somewhat puzzling to analyze

During the past three years so many cases of the kind have been observed that from our past experience we have been enabled to foretell, to a degree, the progress such cases would make. To add to this assurance, lately it has been my privilege to see many patients admitted to the hospital for divers complaints of middle age, in whom we recognized the final compensatory stages of this syndrome. Upon close questioning, their antecedent history bore out the facts that our studied clinical types presented to us in various stages of the syndrome. They were completely compensated cases and their presence in the hospital was for some entirely adventitious cause. Our observation has disclosed clinical types presented by single cases at various ages and stages of the syndrome, progressive cases, observed over periods of from one to six years, which show the changing and probably compensatory nature of the disturbance, completed cases in which the disturbance had come to a definite stop, in which the antecedent history revealed the close relationship to our isolated cases which were still in active progress, uncompleted cases in which the condition, after passing through the preliminary stages, remained indefinitely progressive. It has been impossible, as yet, to observe one case from the beginning to the end of the syndrome—a period which varies from ten to twenty years—and until this can be done we must fill in the gaps as best we may.

GENERAL DESCRIPTION

This new syndrome, pieced together as described above, may be generally stated to begin in youth some years before puberty, and go through its varying stages in about twenty years. In its incipiency (*first stage*) it presents largely the characteristics of the so-called status thymicolymphaticus, or status hypoplasticus of Bartels. There is complaint of muscular fatigability as a subjective sign with frequent accompaniment of headache. Objectively the case presents usually, though not invariably (for

is 6 feet high or over, his weakness, even though his musculature seems splendid, is his prominent symptom. He shaves rarely or never. Pubic and axillary hair remain as before. Now he begins to notice an enlargement of his hands and feet, and a frontal headache or, rather, an intratemporal headache comes on. Blood-pressure remains low (90 to 100 millimeters systolic), blood-sugar usually remains low, but now frequently rises as compensation progresses. Our patient shows decided vagotonic symptoms. An x-ray of the skull at this stage, or during this stage, if the case progresses favorably, shows a sella turcica which, while small, may show erosion of the clinoids and a deepening of the cavity. This tendency of the pituitary to become hyperactive produces the headache (if the sella is contracted), the increase in blood-sugar content, the growth of hands and feet, and a gradually rising blood-pressure.

The *fourth stage* now comes on from three to ten years later. This is the stage in which either complete compensation is produced or else the untreated case takes on the varying and various attributes produced by an enlarged pituitary body engrafted upon the earlier manifestations of a thymic state. That is, we have in the completely compensated case features of acromegaly, although the blood-pressure and blood-sugar are normal and the headaches have gone. The sella turcica on x-ray examination seems large. In the uncompensated cases we usually see a sella which is still small and perhaps bridged, with headaches of increasing severity, perhaps attacks of petit or grand mal, dependent upon the disturbance of pituitary function, mental torpor, increase of weight with constantly increasing fatigue, and a final lethal termination in intercurrent disease.

A brief résumé of the characteristic symptoms and findings in the different stages is here succinctly put forth.

First Stage—The bony structure shows various endocrine anomalies and defects, disproportion of various skeletal units, teeth late and usually characteristically anomalous, so that the lateral incisors are very small, the central incisors disproportionately large, the canines of the type of incisors with a cutting edge instead of a tearing point, epiphyses slow in joining shafts

of bones, hyperextension of joints, hair growth late and sparse, cramps in muscles, tendency to hemophilia and spasmophilia, enlarged thymus, maxillary torus, tonsils large and adenoids present, low blood pressure, low sugar content of blood, epistaxis, cyanosis of extremities, fatigability, small sella turcica, enuresis, low CO₂ coefficient of the blood

Second stage begins at about age of puberty, rapid skeletal growth begins, late menses and small or infantile uterus, great fatigability with all evidences of low adrenal supply—low blood-sugar, low carbon dioxide combining power of the blood, white line of adrenal insufficiency, pubic hair of invert type, lack of hair on face and chin and axilla, smooth soft skin of child, genitals of invert type or else retarded, vagotonia, symptoms of hyperacidity and gastric ulcer, enuresis, undue length of long bones, low blood pressure, little stamina.

Third Stage—Twentieth to thirtieth year, beginning giantism, headaches, pituitary in character, drowsiness, acromegalic beginnings or other pituitary stigmata, fatigability may continue or improve, mental confusion and hebetude, epileptiform attacks, uncinate in type, sella turcica enlarges or else erosion of sella or clinoids takes place, blood-sugar gradually increases if a cure is established. In uncompensated cases mental symptoms, moral and intellectual deficiencies and delinquencies arise

Fourth Stage—Either complete compensation, so that the patient may live comfortably within limits of exertion, or else may progress to the end of life as a pituitary case. The various external manifestations of pituitary disturbance remain even if the physiologic cure is complete. Blood pressure rises, headaches cease, fatigability vanishes.

DISCUSSION OF SYMPTOMATOLOGY

The bony structure in the first and second stages usually shows anomalies in proportionate skeletal growth, i.e., legs too long for thorax or vice versa (my scale for this determination is a fraction with the numerator as the distance from the sternoclavicular junction to the anterior superior spine of the ilium of the same side, and the denominator the distance from the anterior superior

spine to the external malleolus, normally, this fraction is one-half—a larger one meaning too large a torso, while less than one-half represents too long a leg, I call this ratio the torso-leg ratio), the joints are usually hyperextensible and frequently the ligaments are so relaxed that dislocations are easily produced. The extremities can be thrown about like flails, while the teeth are usually delayed in their appearance they also show certain characteristics. The lateral incisors, especially in girls, are frequently greatly underdeveloped. The canines, likewise, are either underdeveloped or else take on the flat appearance of incisors, losing their fang-like appearance. With the cyanosis of the extremities we occasionally get a pustular-like eruption about the nails. The symptoms during the second stage may need some elucidation. The so-called "white-line" of adrenal insufficiency was first described by Sergent. It has not been proved to be due to the deficiency, but in my experience it invariably accompanies low blood-pressure, and may be made to disappear very quickly after a hypodermic injection of adrenalin. Previous emotional disturbance, even so slight as that produced by standing before a camera to be photographed, will make its appearance impossible to obtain. It is obtained best by having the patient lying quietly in bed for a short while and then stroking the skin, preferably of the abdomen or thigh, lightly with the palmar surface of the index-finger. In ten to twenty seconds there will be a blanching of the skin thus stimulated. (See Sergent's article, *Endocrinology*, 1917, 1, 18, for the explanation of this phenomenon.) The low carbon-dioxid combining power of the blood plasma diminishes the so-called "buffer" property of the blood and leads to acidosis on slight provocation. The smooth soft skin of these cases, even in the third decade of life, with little or no secondary hair on the face, a faint suspicion of lanugo on the lip and chin, and a "peaches and cream" complexion, frequently stamps these cases at sight. The headaches, produced as will be discussed in the pathogenesis, are of a specific type. They are invariably stated to be between the temples, the patient indicating the locality by putting one index-finger on each temple, directed mesially. We have come to call them "pituitary headaches."

The vagotonia present in many of the cases frequently takes on the character of hyperacidity of the gastric juice with frequently symptoms of gastric ulcer, spastic constipation, and eosinophilia. In the third stage the symptoms of great interest are the mental ones. In many years' observation of pituitary disorders we have been frequently struck with the mental quirks of the hypopituitary. He exhibits lack of inhibition of the emotions, becomes highly excitable on little cause, alternating with sluggishness, frequently has phobias and compulsions (one case was a true kleptomaniac), shows frequently moral and sexual obliquities, and exhibition of pituitary feeding often modifies these characteristics. The symptoms of the uncompensated cases usually merge into those of a frankly dyspituitary syndrome. Blood-sugar disturbances, intense fatigability, periodic headaches, temperamental unfitness, and drowsiness are among the prominent symptoms.

ETIOLOGY

In practically all our cases there have been family histories of importance as regards endocrinopathies. Frequently parents or grandparents have shown such disturbances as diabetes, goiter, or acromegaly. A very common complaint is giantism. Collateral branches, too, show similar disturbances. Thus, W W (Fig. 190), the fully compensated case, has four cousins all afflicted with Graves' disease. There appeared in our cases no particular antecedent disabling disease or injury. One case, now in the second stage, had two brothers, both dying suddenly after exertion without known cause, in youth—possibly a so-called thymic death. Periodic headaches are also distinguishing marks etiologically. Menstrual disturbances of all kinds are met with here, especially the late appearing type with lack of periodicity.

DISCUSSION OF PATHOGENESIS

During the first stage we see a clinical picture which is dominated by the characteristics of the *status hypoplasticus* of Bartels. The anomalies have been variously credited to hypo-function of the individual endocrine glands, excepting the thymus, which is supposedly hyperactive. Thus, Tandler and Gross

and Tandler have described many of the features of such a condition due to deficiency of the gonads. And yet, in direct contradiction to their view that gonadal deficiency produces growth in height with late joining of the epiphyses, I have seen cases in which at the age of eighteen with no menstrual flow yet established, the sexual apparatus quite infantile, the α -ray of the long bones showed the epiphyses almost united, the height of the patients being under 5 feet. Wiesel, Schur, and Schmorls and Ingiers have given both clinical descriptions and histologic and pathologic findings in such hypoplastic conditions referable to underactive or inhibited adrenal glands. Many observers have described the smallness of the sella turcica. My own observations agree with these. All of our cases show the smallness of the sella turcica in the early stages and, in addition, many of them have the bridging over by the clinoid processes, evident on α -ray examination. With these deficiencies of glandular structure and their diminished physiologic activity *ab initio*, the organism would of necessity come to early grief if some corrective were not forthcoming. Many cases do succumb early. Undue exertion, sudden excitement, narcosis, are all critical moments for such organizations, many of which cannot survive them. After puberty should have been reached (the second stage) the deficiency of the gonadal inhibition to growth (Tandler and Gross) is claimed to be responsible for the extreme height rapidly reached by our cases. One of our cases (Private B), however, rather opposes this theory, in that the gonadal system early became hyperplastic and still the growth in body took place. This excessive genital development may be due in his case to an early involution of the pineal gland, for in the α -ray calcification of the pineal is seen. (Some authors hold that the overactive thymus with disturbed calcium metabolism is the cause of such "thymic giantism".) The deficient adrenal-chromaffin system is to be credited with the great fatigability, the low blood-sugar content, the low blood-pressure, and the white line. Now comes the third stage, the all-important one. It is in this period that the outcome of the syndrome is determined. In our judgment it is the pituitary gland which is here the critical factor. As we have seen, it

is invariably enclosed in a small sella turcica and possibly even hemmed in by the clinoids. Among its functions we have as all important a blood pressor principle and a sugar mobilization factor. Both of these are deficient in our patient. If the pituitary possibly could become hyperplastic and hyperactive with an intensification of these important properties, compensation might be accomplished. Such tendency to hyperplasia in a small cavity would of necessity through pressure produce headaches—an invariable symptom in the third stage of the compensated cases. And such headache would continue until the enlarged gland through erosion of its bony capsule or through pushing apart the clinoids made sufficient room for itself. As will be seen, these headaches continued for two to three years in some of our cases. Synchronously with these headaches, other incidental features of an enlarged pituitary gland became manifest: (a) acromegaly, lasting until the headaches ceased and the process then likewise ceasing, (b) a higher blood-sugar content, (c) a higher blood pressure, (d) a diminished sugar tolerance. To make this view of the nature of the process of compensation more tenable many of the sellæ turcicæ of our patients in the second and third stages show erosion of the anterior or posterior clinoid processes, and in the final stage, an enlarged sella with practically no clinoid processes left at all. In the cases in which no compensation was effected—*e*, in which fatigability and the other symptoms remained and progressed—the sella showed no enlargement (notably that of T. R., Fig. 192). In these cases we had headaches, periodical in type, adiposity, mental and moral deficiencies, petit mal, and other manifestations. Curiously enough in all our cases, the feeding of the pituitary gland in fairly large quantity disposed of many and at times of all these symptoms. But if the feeding were diminished or stopped, the symptoms reappeared. It seemed analogous to thyroid feeding in myxedema. One case, which gave a typical early history and seems uncompensated today at the age of forty-four, still shows the very small sella turcica with a clinical picture of abnormal bony structure much resembling Paget's disease. On pituitary feeding, this case is improving markedly in its fea-

tures of fatigability, headaches, and heaviness of extremities. It is too early to state whether in her case the cancellous condition of the bones will be restored. Finally, the fourth stage is ushered in by a gradual cessation of the fatigue, amelioration of the headaches, restoration of a normal blood-pressure, and normal sugar content of the blood. But the adventitious signs of the disturbance of the pituitary gland remain. Thus the fully compensated cases may show acromegaly more or less marked, *and this acromegaly is not to be taken as a diseased condition needing treatment, but simply as the hallmark of a process that has come to a stop—a self-curable process.* It is analogous to the hypertrophied heart become so through the deficiency of the cardiac valves and making up for such deficiency by its enlargement. And that condition likewise, *per se*, needs no treatment. A case that presents acromegalic features, therefore, need not necessarily be a case that calls for therapeutic intervention. It may well be a "finished" case. W W (Fig 190) is a good representation of this type. These "finished" cases must always, however, live within certain limits of exertion and stress. The cases that in the fourth stage do not spontaneously go to full compensation are those in which we either find a sella turcica which did not enlarge (perhaps because there was no spontaneous effort of the pituitary to become hyperactive) or in which an enlargement of the sella did take place and the pituitary even in its hyperactive condition was not sufficient to compensate. These uncompensated cases go right on with progressive symptoms of fatigability, asthenia, headaches, and so forth, making them easy prey to intercurrent affections.

TREATMENT

The treatment of these cases in any stage is extremely satisfactory. The great point to remember is the probable nature of the process of compensation which the organism is endeavoring to carry out. That would make one believe that suprarenal gland therapy is indicated throughout on account of the patent deficiency of this organ in these cases. And yet in our hands its administration is disappointing. The whole gland perhaps has given better results than adrenalin, although the latter, either

hypodermically or (even against the dictum of the physiologist that it is inert when administered per os) by mouth in larger doses is good to tide over exceptionally bad days of fatigue and exhaustion. But the prime agent—almost a specific one—is pituitary gland in some one of its varied forms. Whole gland feeding in fairly large doses (2 to 3 grains t. i. d.) may be given in appropriate cases. But usually the dosage should be much smaller, and given at greater intervals, $\frac{1}{2}$ or $\frac{1}{4}$ grain every second or third day has given success in several patients. Occasionally, pituitrin hypodermically 0.50 to 1.00 c.c. per day or alternate days for one or at most two weeks at a time is excellent as supplementing the feeding of pituitary gland. Occasionally, in cases with pronounced genital delay, anterior lobe pituitary gland gives fair results. In those cases with vagotonic symptoms, hyperacidity, and conditions resembling gastric ulcer, atropin in doses to physiologic tolerance is indicated, and gives results. But the pituitary feeding in itself alone produces highly satisfactory improvement in almost every case. Under its use the headaches disappear, the fatigability diminishes, the blood pressure and blood-sugar content increase, and the case goes on to cure. Gradually the pituitary feeding can be diminished and finally discontinued. In the older cases, in which the sella persists in remaining small (T. R., Fig. 192, and J. S., Fig. 191), constant feeding would seem to be necessary, at all events, the patients relapse as soon as treatment is stopped. Indeed, the patients themselves reach that point of accuracy of judgment in feeding the gland to themselves that they can determine the size and frequency of the dosage necessary to maintain them comfortably.

CASE HISTORIES

The following case histories are cited as a few among many which show characteristics of the different stages of the syndrome. Only facts having direct bearing are introduced into the case histories.

CASE I—J. M. (Fig. 181) Ten years, mentally backward, no hair on body anywhere, genitals undeveloped, elongated thorax, spasms in muscles, great fatigability, is a bed wetter,

joints are hyperextensible, has nosebleeds τ -Ray of chest shows thymus (Fig 182), skull shows small sella turcica, entirely bridged over (Fig 183) Blood-pressure 85, white adrenal line

Discussion—This case is presented simply to show the type from which arise the cases presenting the later features of the syndrome, and hence may be classed under stage one



Fig 181—J. M. Case I. Shows abnormal length of thorax compared with legs. Thighs especially short compared with lower leg. Small genitals. Has large thymus and enclosed sella turcica.

CASE II—Master F. J. Age thirteen and one-half years, height, $61\frac{1}{2}$ inches. He was brought to the Neurological Institute on account of his predilection for lying and, further, for his rapid fatigability. He had been discharged from several schools on account of his incorrigibility. The climax was reached when he appeared as a material witness in a murder trial, his evidence

being of the greatest importance in the conviction of the accused. Apart from these facts, he also complained of spasms in the muscles, especially of the calves, which occasionally awakened him by the pain caused thereby. He had frequently also had nocturnal enuresis. Upon stripping him (Fig. 184) the examination revealed the following—the body was perfectly hairless, no trace of pubic or axillary hair being evident. The genitals were small



Fig. 182.—J. M. Case I. Sella turcica small and entirely enclosed and roofed in.

—infantile in fact—but both testicles were descended and the scrotum surrounded the penis like a labial fold. Upon eliciting the cremasteric reflex, however, the testicles were drawn up out of the scrotal sac into the inguinal canal, where they could not be palpated. He bruises very easily, the dark patch on the left raised arm being produced by the simple pressure of the examiner's thumb in raising the arm. Hands and feet are somewhat larger than they should be. Stroking the skin of the abdomen

produced a marked white line of reaction, which persisted for a rather long time. His blood-pressure was low—80 mm. Mentally he was well up to his chronologic age according to the Binet-Simon scale. Upon close questioning he admitted that he had frequent headaches referable to a point midway between the temples and deep seated

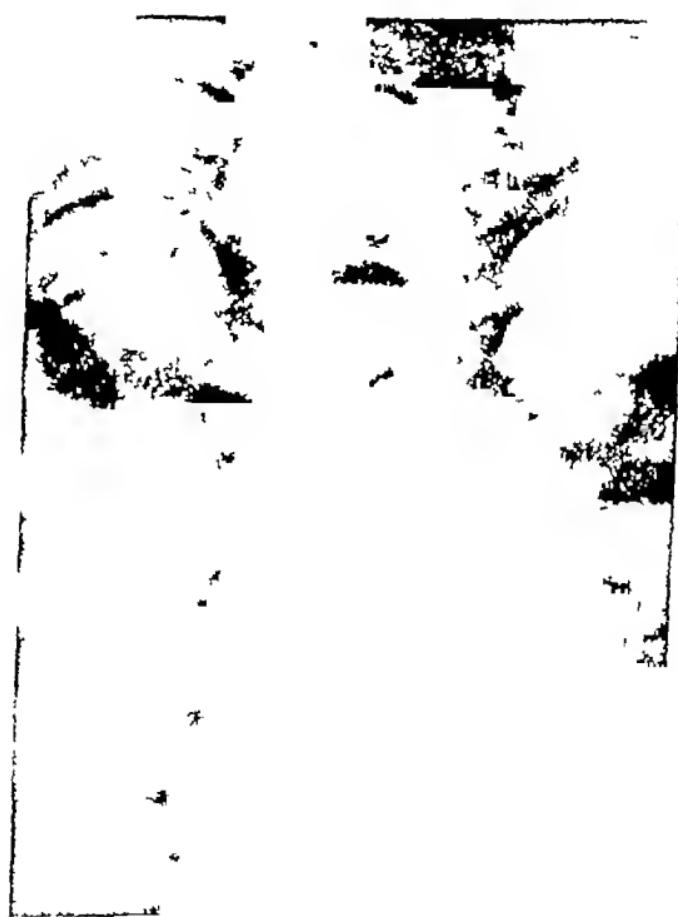


Fig 183—J. M. Case I. Shows thymic enlargement

The neurologic examination was negative. Laboratory examination showed the sugar content of the blood to be 0.070 per cent. Otherwise blood and urine were negative and the Wassermann was negative. α -Ray examination. The sella turcica (Fig 185) was small and encroached upon by large chnoids, the upper thorax showed a thymus shadow.

Discussion.—This case is presumably a type of the beginning of the second stage of the syndrome. It presents many of the well marked features of a *status hypoplasticus* small sella, enlarged thymus, low blood pressure, low blood-sugar, marked fatigability, spasmophiliac and hemophiliac attributes in addi-



Fig. 184.—F J Case II Age thirteen and a half years. Shows hypoplasia of genitals and a scrotal fold surrounding base of penis. Absence of hair. Bruise on raised arm from slight pressure

tion to the obvious externals. The mental attributes are those which we frequently see in small and enclosed *sellae turcicæ*. This case is presented simply as a living present example of the early beginnings of the second stage of the syndrome.

CASE III.—Private B (Fig. 186) Age twenty years, height 6 feet 1 inch. This case was sent to the Institute by Capt.

Reed, stationed at Fort Wood, to determine whether or not the man was a malingerer. His one complaint was that of excessive fatigability after moderate exertion. As a result he could not perform the military duties required of him. His musculature and his whole bearing and appearance when stripped were that of a powerfully and symmetrically built young giant,



Fig. 185—F. J. Case II. Pituitary fossa shut in extremely by large chondrocytes.

and seemed to belie his statement of rapid fatigue. His early history could not be obtained with any degree of reliability, for, coming from a mountainous district of the South, he was extremely uncommunicative. In early adolescence he was a rover, traveling over many states in divers occupations, but never steadily at any one. He did say that for the past three years he had

been rapidly and steadily growing in height, that his growth was still continuing, and with it his fatigability was increasing. Stripped, he showed a splendid make up physically. Of note was the fact that he had practically no hair on his lip or chin, no hair in the axillæ, pubic hair of the feminine type, and rather largely developed genitals. Stroking the skin produced a white



Fig 186.—Private B Case III Feminine distribution of pubic hair large genitals height 6 feet 1 inch No hair on face. Seems well proportioned

persisting reaction. His blood pressure was between 95 and 100 mm systolic, 80 diastolic. Blood sugar was 0.062 per cent. x Ray examination showed a sella turcica (Fig 187) which was extremely small, with thickened anterior and posterior clinoid processes completely roofing in the cavity. A shadow in the pineal region also was evident. The thorax showed a thymic

shadow. Neurologic examination was negative. Viscera appeared normal.

Discussion—This young man is going through the transition period from the second to the third stage. His low blood-pressure, low blood-sugar percentage (extreme normal low limit



Fig. 187.—Private B Case III Complete closing in of pituitary fossa by large chondros

should be at least 0.075 per cent), white skin reaction line, all go to prove his statement of fatigability. In addition, he shows a crowded pituitary fossa and a pineal shadow. We have found in the past two years in a large percentage of muscular dystrophies, pineal shadows, and in extreme cases of muscle fatigue in the

adolescent, short of dystrophy, there were shadows in the pineal region. This parallelism between the myasthenic types and early involution of the pineal gland will be the subject of a future paper. This feature of the case is an example of many similar ones. But, presumably, early pineal involution also produced his enlarged genitals. He is undergoing the rapid growth incident to the second stage of the syndrome in spite of the fact of the enlarged genitals. Some authors (especially Tandler and Gross) have maintained that the hypoplasia of the genitals accounts for the continuance of the skeletal growth. That is certainly disproved here as a universality. Upon feeding of pituitary gland this youth improved sufficiently to go back to army life. After the lapse of a few months he discontinued this feeding, and shortly thereafter returned with his old complaint of fatigability. He is again improving on treatment. No white line can now be elicited, and his blood pressure is usually at 120 mm.

CASE IV—G. E. R. (Fig. 188) Age twenty two years, height 6 feet, $3\frac{1}{2}$ inches, single, stenographer. Came to the Neurological Institute complaining of loss of memory and lack of sleep, feeling stuporous and extremely fatigued. His head aches intratemporal in location. Not able to do much work on this account, and hence took a position as stenogra-



Fig. 188.—G. E. R. Case IV
Height 6 feet, $3\frac{1}{2}$ inches feminine
pubic hair no hair on face muscula-
ture seems flabby feminine attributes.

Is exempt from military duty, being the sole support of his mother

Past History—While a child he was never strong, he took a long time to develop, and on account of his ways—feminine—was always known as “sissy” to his playmates. He realized this appellation to be more or less just. Would easily cry if deprived of his way, and still does so. Always was a bed-wetter, and this weakness persisted to his nineteenth year. Realizing his fem

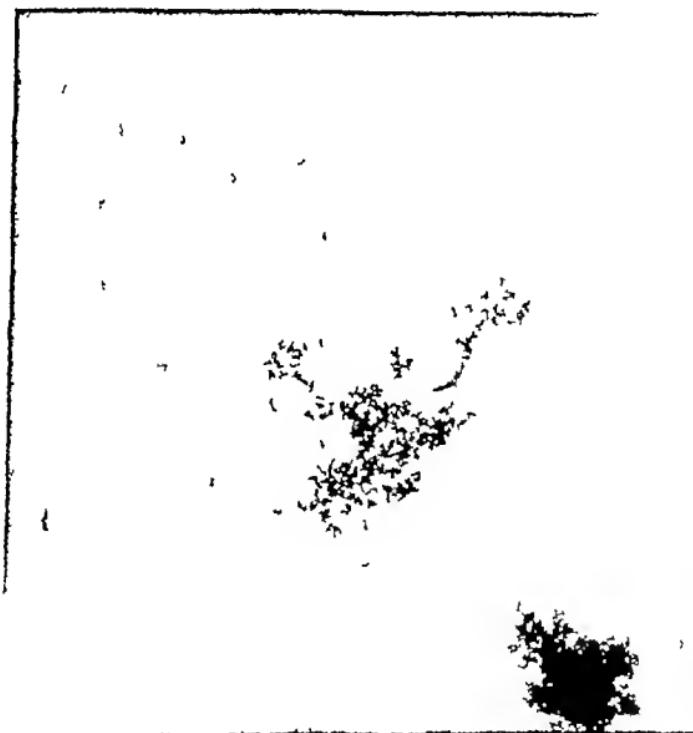


Fig 189—G E R. Case IV. Sella turcica with anterior cavity eroded and a deepening of posterior fossa.

inine attributes, he endeavored to compensate for them by indulging in the most manly of sports—boxing and football. But he was too slow to amount to anything here. He gave up this conflict and sank to his own recognized level—became a stenographer. He began to get headaches at seventeen years of age, at which time his excessive growth started.

Examination—A man powerfully set-up, 6 feet, $3\frac{1}{2}$ inches high and proportionately built (Fig 188). No hair on face or

body except in the pubic region, where it is of feminine distribution. The scrotum also is divided above the penis into typical labial folds. He shaves once in two weeks. His appearance is really pugnacious, but is not borne out by his mental attitude. He cries when questioned about the simplest difficulties. His blood pressure is 100 mm., systolic. He has the white line reaction of the skin, and his blood-sugar was low. The x-ray of the skull shows a sella in which erosion of the anterior clinoid is evident and an excavation of the posterior portion of the floor (Fig. 189). The x-ray of the thorax shows a possible enlargement and persistence of the thymus. Neurologic examination is negative.

Discussion.—The early history is that of a thymus state. The further progress, especially the headache concomitant with the growth, is suggestive of pituitary enlargement. This is partly borne out by the picture of the sella. His headaches are now much less prominent than they were, but his fatigability is marked. His growth seems to have ceased. This case belongs in the third stage—with the giantism gradually reaching its acme, with the stuporous and drowsy mental condition of a dyspituitarian still present, great fatigability, low blood pressure, and low blood sugar content. The enlargement of the sella turcica, however, gives promise of a gradual efficient compensation taking place. Feeding of whole gland pituitary extract within two weeks measurably improved all his symptoms. It is still being continued.

CASE V—W. W., thirty two years, machinist, married, several children, height 6 feet, 4½ inches, acromegaly in slight degree. This case has been under our observation since 1912. At first the complaint was one of utter exhaustion to the exclusion of all minor ills. He could not stand at his work, he could hardly walk without becoming exhausted. At nineteen he had severe headaches referred to the intratemporal region, which persisted in spite of all measures. These headaches he described as "crushing." At the same time his hands enlarged while under our observation. The fatigability was so great that he would have to take to his bed and remain there. His past history was typical of the thymic. Nosebleeds, general weakness, enuresis,

overextensibility of the joints. His family history was a typically endocrine one. On his mother's side four members of direct and collateral branches had goiter, two with exophthalmos. His father never shaved until the age of twenty-eight. Puberty in



Fig. 190—W. W. Case V. 6 feet, 4½ inches. Fully compensated case. Great length of leg compared with length of thorax. Feminine waist, feminine pubic hair, acromegalic hands, no hair on face.

this patient was reached at nineteen. Hairy growth in the axilla and on the face remained absent. To this day he has never shaved. During the period of intense headaches and growth he had various vagotonic symptoms, hyperacidity, excessive perspiration, precordial distress, nausea and vomiting, marked pallor, lack of libido. His gastric distress became so acute that operation was advised at another hospital for gastric ulcer. At this time an α -ray of the skull was taken and showed a large sella turcica, with practically no chondoids present at all, and erosion at the edges. His photograph (Fig. 190) has some interesting points. The extreme length of leg compared with thorax is noteworthy. The feminine distribution of pubic hair and feminine waist are present. His extremely large hands and generally deficient musculature attract notice. This photograph was

taken about a year ago when he was just completing his cure. Today he has gained 30 pounds in weight and is feeling practically well, working constantly as machinist, and supporting his family of wife and two children. During the last three years his blood-pressure had gradually risen to 130 mm. systolic from an initial

90 to 100 systolic, his gastric symptoms have abated, the white line of adrenal deficiency is disappearing, his fatigability has passed, libido has returned, and he is, to all purposes, cured of his malady. His fatigability and headaches in the past three years could always be improved with the administration of whole gland pituitary extract, and gradually it became possible to diminish the dose and to allow the compensation to proceed unaided. He has now been without treatment for practically six months. He seems to be a fully compensated case.

Discussion.—The history and progress of this case is typical of all cases passing through the complete syndrome, and is presented as a type of which I have now seen at least ten examples. It must not be forgotten that he now shows giantism plus acromegaly. That does not mean that his clinical picture indicates treatment. Far from it. His is a finished case. The giantism and acromegaly are incidents merely of the compensatory process—they themselves do not necessarily indicate present disease. This is a point well worth remembering. Many cases showing endocrinopathic features come under our notice, and it should be our first aim to determine whether or not they represent processes that have come to a stop through compensatory efforts of which they are the indices. Under such conditions, of course, no treatment is called for. An interesting case of this nature was seen at Mt. Sinai Hospital, New York, in the service of Dr. Goldenberg, through the kindness of Dr. I. Strauss. A woman, fifty three years of age, with typical acromegaly—hands, feet, and skull abnormalities—was admitted for some minor ailment. Her history showed that she began to menstruate at nineteen years, had intense headaches at twenty five years, which for three years resisted all attempts to alleviate, and during whose persistence she began to grow acromegalic. When the headaches spontaneously ceased the abnormal growth ceased, and she passed an uneventful life to the time of the present slight ailment. Her childhood and adolescence were like those described in the first two stages of this syndrome—fatigability being the prominent symptom. Her acromegaly then was completed at about the twenty ninth year did not further increase, and she remained

well for twenty-three years longer. This was also a "finished" case as far as the syndrome of thymus, adrenal, and pituitary was concerned. This case might be many times duplicated.

UNCOMPENSATED CASES

I desire to give rapidly the salient features only of several uncompensated cases belonging to the syndrome.

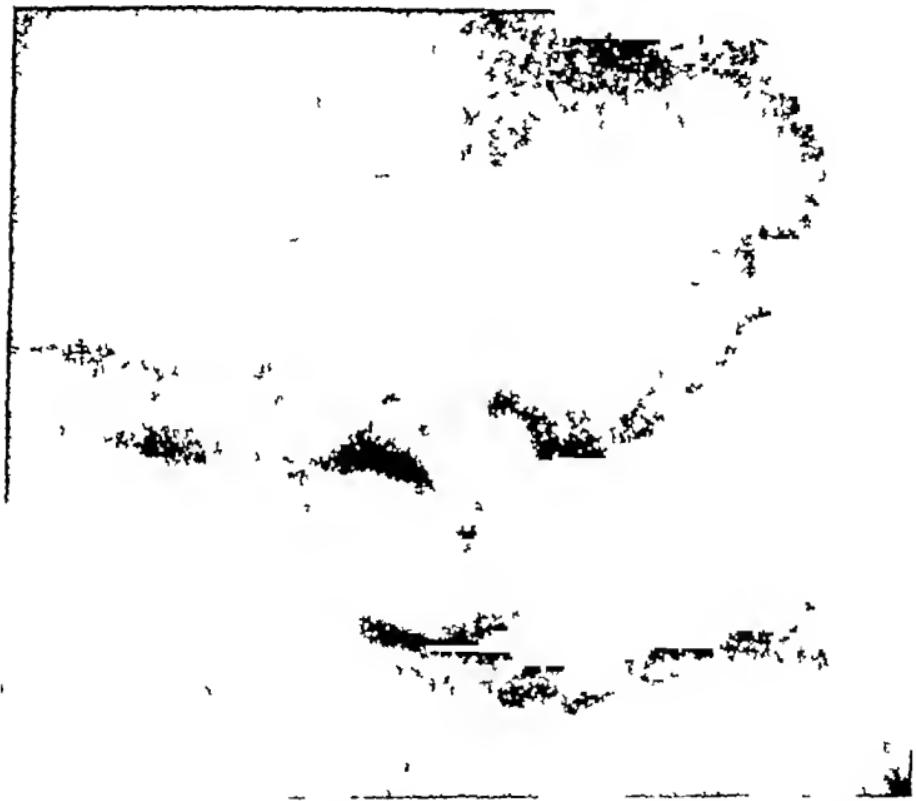


Fig 191—J S Uncompensated Case 186 Shows pituitary fossa completely enclosed. Stereoscopic plates confirm this. Age twenty-two years, height 6 feet.

CASE VI—Miss J S. Referred by Dr Robert T Morris. Height 6 feet, large hands and feet, extreme fatigability, headaches, vagotonia, rapid growth in past two years, with a past history of status thymicolymphaticus, comes for relief of her headaches and fatigability. Blood-pressure 95 systolic, blood-sugar 0.065 per cent, fatigue so great that she must remain prone the greater part of the day. Intense white line. Her sella turcica

(Fig 191) shows a roofing in by the clinoids. Pituitary feeding gave gratifying results, but must be continuously carried out.

CASE VII—T R Age thirty five years, height 6 feet, 1 inch. Obese (220 pounds) Early fatigability Late puberty Feminine pubic hair Unmoral Intense intratemporal headaches Although wealthy, and of excellent family, commits sexual and other excesses Frequently in jail Fatigability and



Fig 192—T R. Uncompensated Case 187 Age thirty five years height 6 feet, 1 inch Sella turcica completely shut in and ~~extremely~~ Confirmed by stereoscopic plates.

CONCLUSION

The syndrome of thymus—adrenal—pituitary combination is one frequently met with, and its various stages are easy of recognition. The main characteristics of fatigability—low pressure, headache, and growth—are invariable components of the syndrome, and depend upon maladjustments of endocrine interactivity. Stabilization of the balance may be spontaneously produced providing the sella turcica may be made to accommodate a hyperactive hyperplastic pituitary gland. This is done presumably by erosion of the bony capsule of the gland. In cases of inability of such enlargement of the sella, the syndrome persists, but the symptoms may be alleviated by the feeding of pituitary extract continuously. In the course of the syndrome other glands may be brought into the complex and alter the picture somewhat, but these are vagaries and seemingly have no great determining effect upon the course of events. Once recognized in any of the early stages, the further general progress of these cases can be prognosticated with a great degree of accuracy, and intervention, if necessary, can be undertaken with a large degree of success in the amelioration of the distressing symptoms.

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Cushing The Pituitary Body and its Disorders Especially Case XXXII—giant Turner, this case seems to fit into the above syndrome almost perfectly, lack of hair, small genitals, low temperature, minute adrenals, intense muscular fatigability, growth beginning to be excessive at age of fifteen, accompanied by intense headaches Cushing himself states that "the patient is not an acromegalic giant" in the true sense (p 168)

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CLINIC OF DR WALTER W PALMER

PRESBYTERIAN HOSPITAL

PNEUMOCOCCUS ENDOCARDITIS

Acute Endocarditis Occurring in Course of Acute Lobar Pneumonia Treated with Antipneumococcus Serum, Early Diagnosis, Care in Administration of Serum

March, 1919

CERTAIN interesting features in the course of the use of antipneumococcus serum in the treatment of acute lobar pneumonia have come to light in the past few weeks, and I wish to present 3 illustrative cases. All of you are now familiar with the biologic classification of the pneumococcus and the importance of this differentiation in the prognosis and treatment of the disease. It might not be out of place for you to read again the clinic by Dr Rufus Cole which appeared in the New York number of the Medical Clinics of North America, November, 1917. Dr Cole describes in detail the manner in which the various types are determined, and also points out that antipneumococcus serum has proved of value in the treatment of cases infected with Type No I pneumococcus. The administration of foreign serum, such as horse-serum, to human individuals must be performed with certain precautions to avoid the production of anaphylactic shock, which at times is fatal. Great emphasis is placed on the manner in which serum should be given. It is not the purpose of this clinic to discuss these details, nor do I propose to dwell on the subject of the general treatment of pneumonia. We will now proceed to a presentation and discussion of the cases.

CASE I (Chart I, Hospital No 37,461) — J. J., age twenty-eight, Spanish cigarmaker, entered the hospital for the first

time February 27, 1918, complaining of pain in left side, fever, and general malaise of six days' duration. His family and past history contain nothing relevant to the present illness (Fig. 193).

Six days ago the patient was seized suddenly with a sharp pain in left chest, followed by a chill, which in turn was followed by fever and cough, causing him to take to bed. In twenty hours he felt much better and got up. Five days later, or twenty-four hours ago, he was seized suddenly with a second chill, pain

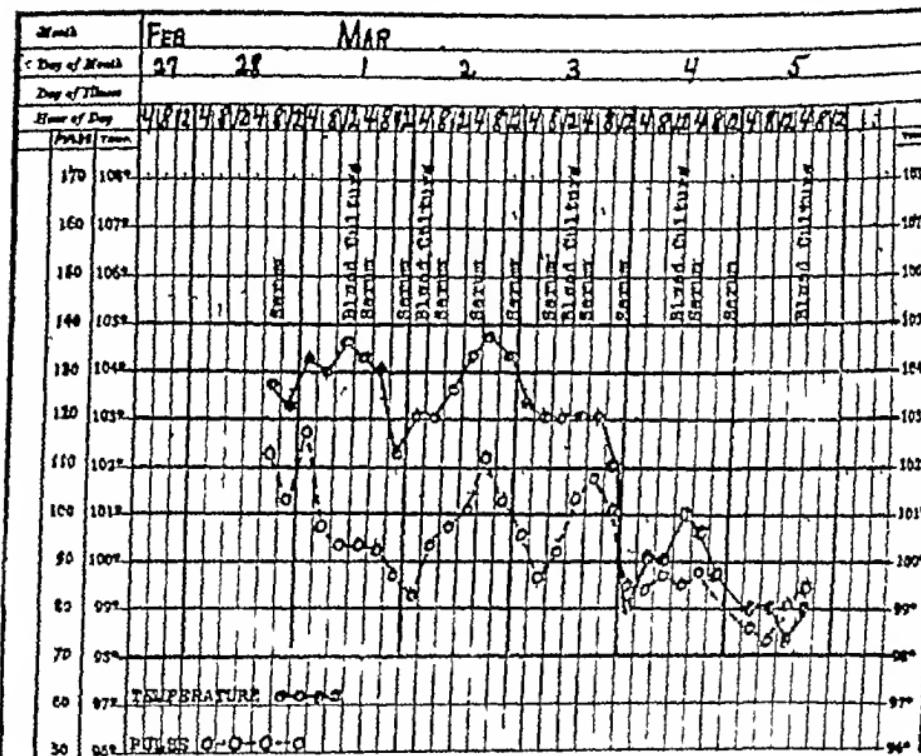


Fig. 193.—Chart of Case I

in the left chest, and was nauseated. Following the chill he became feverish, much prostrated, a severe headache developed, and he began to cough up bloody sputum. Deep breathing and cough produced much pain in the left chest.

The physical examination presents little of especial interest except in the left lung posteriorly, where from the lower angle of the scapula to the base there is dulness, increased voice whisper, a few crepitant râles, and friction-rub. W. B. C. 12,000, 87 per cent polynuclears. Urine showed nothing abnormal.

No difficulty presents in making the diagnosis of acute lobar pneumonia. It is of interest to pause a moment on the history. Are we dealing with a one-day or a six-day-old pneumonia? The history is very suggestive of a one-day pneumonia, occurring six days ago, with a more definite and severe attack twenty-four hours ago. As it is not possible to be sure on this point, we shall have to leave it an open question.

On the day of his admission, as usual, the sputum was sent to the laboratory, but through some error the typing was delayed, and it was not until the second day after admission when the blood-culture revealed a Type I pneumococcus, 1-2 colonies per c.c., that the type of infection was known. The same day a Type I pneumococcus was obtained from the peritoneal washings and heart's blood of a mouse which had been injected with the patient's sputum. At this point let me emphasize the great importance of determining the type of pneumococcus at the earliest possible date. I could present case after case where serum has apparently shortened the disease by several days. In certain of the cases of lobar pneumonia occurring among the hospital staff the diagnosis of type has been made, the patient desensitized, and serum given within twenty four hours after the initial chill, and I have seen the temperature come to normal after one intravenous treatment of antipneumococcal serum.

February 28th The usual intracutaneous injection of 0.02 c.c. of a 1:10 dilution of normal horse-serum was made without evidences of hypersensitivity. One hour after this test was performed the customary $\frac{1}{2}$ c.c. of horse-serum to desensitize the patient was injected subcutaneously. Within ten minutes the patient became moderately dyspneic and restless for a period of about one minute. One hour and a half later preparation for injection of the usual 100 c.c. of antipneumococcus serum Type I was made. About 1 c.c. of the serum was injected, taking about five minutes, when suddenly the patient began to cough violently, sitting up in bed, with a severe choking sensation. The injection of serum was immediately stopped, and 10 minims of adrenalin solution, 1:1000, injected subcutaneously, with almost immediate relief. If any of you in the future contemplate the

use of serum intravenously, I trust you will keep well in mind the above incident. The symptoms following the injection of $\frac{1}{2}$ c c of horse-serum subcutaneously should have been a warning of the sensitiveness of the patient, and the desensitization process so carefully described by Dr Cole and co-workers resorted to. In this instance enough serum has been given apparently to accomplish this, for the day following we note

March 1st, 1 30 P M "2 c c of half horse-serum and half saline was injected intravenously. No reaction. Injected very slowly with adrenalin at hand."

2 15 P M 90 c c antipneumococcus serum injected intravenously. No reaction

Before the intravenous serum of this day a blood-culture was taken, showing an average of 900-1000 colonies of Type I pneumococcus per cubic centimeter

	11 00 P M	100 c.c antipneumococcus serum intravenously
March 2d,	9 00 A M	100 c.c. antipneumococcus serum intravenously
	5 00 P M	Blood-culture—sterile
	6 00 P M	100 c.c. antipneumococcus serum intravenously
March 3d,	12 30 A M	100 c.c. antipneumococcus serum intravenously
	10 30 A M	100 c.c. antipneumococcus serum intravenously
	4 00 P M	Blood-culture—sterile
	4 45 P M	100 c.c. antipneumococcus serum intravenously
March 4th,	12 30 A M	100 c.c. antipneumococcus serum intravenously
		Blood-culture—sterile
	2 30 P M	100 c.c. antipneumococcus serum intravenously
March 5th		Blood-culture—sterile
March 8th		Serum sickness

The patient made an uneventful recovery, with no extension of the process after the initiation of the serum treatment

Two important facts are strikingly illustrated by this case
 (1) Great care must be exercised in the administration of anti-pneumococcus serum, (2) serum sterilizes the blood in an uncomplicated case. It is necessary to draw conclusions with great conservatism, but we cannot help but be impressed by the fact that on the day of entrance there was only one to two colonies per cubic centimeter of blood, and two days later the bacteria had increased to 900 to 1000 colonies per cubic centimeter of blood

We know from considerable clinical experience that an increase in the bacteremia in pneumonias is of bad prognostic omen. In fact, a bacteremia of over 100 colonies per cubic centimeter carries with it a definite bad prognosis in the average case. The prompt sterilization of the blood by the administration of antipneumococcus serum is certainly convincing evidence of its value in cases such as the one under discussion.

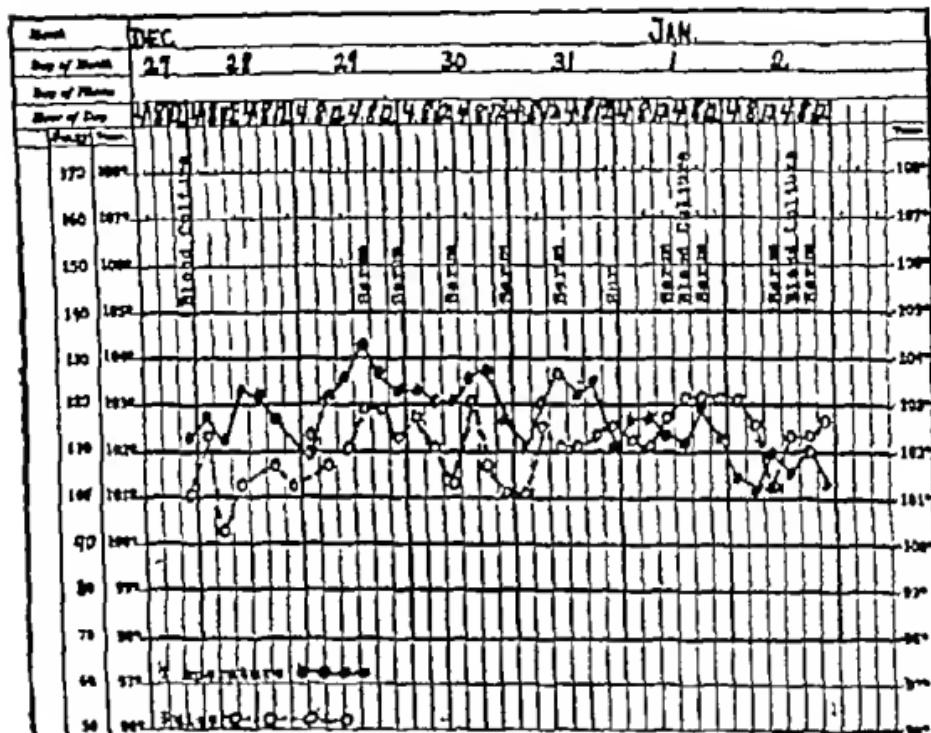


Fig. 194.—Chart of Case II

CASE II (Chart II, Hospital No. 36,934)—G. H., an American printer, aged fifty five, entered the hospital December 27, 1917, for the first time, complaining of pain in the right side, difficulty in breathing, and general weakness of five days' duration. His father and mother both died of pulmonary tuberculosis. The patient is subject to colds, with hoarseness and cough, frequently requiring a day or two in bed, but has never had pneumonia. Never had rheumatism, denies syphilis (Fig. 194).

Five days ago, quite suddenly, felt chilly and much prostrated, immediately taking to bed. The day following there appeared in the right lower axilla pain, which was increased on cough and deep breath. He began to cough and raise bloody sputum. Both cough and pain have persisted, and for the past two days there has been difficulty in breathing.

The patient is a poorly nourished old man, lying quietly in bed, cyanotic, breathing rapidly and with difficulty. There is extensive herpes of the upper lip. The left side of the chest moves with greater excursion than the right on deep inspiration. On the right side, from the lower angle of the scapula to the base, there is dulness, diminished tactile fremitus, bronchial breathing and voice, with many crepitant râles. At the base in the posterior axillary line is a friction-rub. The left lung presents no adventitious signs.

Heart Apex-beat not seen or felt. No thrills. The heart sounds are regular and of fair quality. No murmurs. The aortic second sound is accentuated. Percussion outlines

1—25
25—2—5
4—3—7
5—4—9
5—105

The pulses are equal and regular, good volume and tension. Artery walls slightly thickened. White count, 15,700, 90 per cent. polynuclears.

The urine is amber colored, specific gravity 1018, containing a trace of albumin, many hyaline and granular casts.

As in the previous case, we experience no difficulty in the clinical diagnosis—that of acute lobar pneumonia. I have given the physical signs of the heart as found at entrance because of subsequent developments. There is no evidence of valvular disease nor has the patient had rheumatism. During life syphilis was not ruled out. Although it is a custom of the clinic to have a Wassermann test on all admissions, for some reason it was omitted in this case. It was later demonstrated

that there was no syphilitic infection of the heart valves. The blood-culture taken on the day of admission revealed a Type I pneumococcus in both broth flasks. As in all individuals with lobar pneumonias who enter this hospital, the patient was tested for horse-serum sensitiveness by the intracutaneous injection of 0.02 c.c. of horse-serum diluted to 1:10. As no reaction followed this procedure, a desensitizing dose of $\frac{1}{2}$ c.c. of horse-serum was injected subcutaneously, also without reaction.

At 6:00 P.M., December 29, 100 c.c. of antipneumococcus serum Type I was injected intravenously. Blood plates made just before the serum was injected revealed 3 to 5 colonies per cubic centimeter. It has been found from considerable experience that such serum administration invariably sterilizes the blood after a single injection—unless, however, there are complications. Keeping this fact in mind, the subsequent record will be of interest.

December 30th	12:15 A.M.	Blood plates showed 35 colonies per cubic centimeter Type I pneumococcus.
		100 c.c. antipneumococcus serum Type I given intravenously
	12:00 M.	Blood-cultures just before serum administration grew Type I pneumococcus.
		100 c.c. antipneumococcus serum intravenously
December 31st	10:00 P.M.	100 c.c. antipneumococcus serum intravenously
	12:00 M.	Blood-culture, Type I pneumococcus.
		100 c.c. antipneumococcus serum intravenously
	10:00 P.M.	100 c.c. antipneumococcus serum intravenously

The process in the lungs remains about as at entrance. There is no evidence of a spread discovered. The patient's general condition is fair.

1918.

January 1st	12:00 M.	Blood-culture pneumococcus Type I
		100 c.c. antipneumococcus serum intravenously
	4:00 P.M.	Blood-culture—contaminated.
	9:00 P.M.	100 c.c. antipneumococcus serum intravenously
January 2d	2:00 P.M.	Blood-culture, pneumococcus Type I 2 colonies per cubic centimeter
		100 c.c. antipneumococcus serum intravenously
	9:00 P.M.	100 c.c. antipneumococcus serum intravenously

The patient appears slightly better this morning Pulse good, and temperature by rectum 100.8° F Signs of resolution are to be made out over the process in the right upper lobe. Posteriorly on the left from the lowest angle of the scapula extending downward and toward the spinal column there is dulness, bronchial breathing, and crepitant râles

White blood count, 22 600, polynuclears, 96 per cent

In view of the fact that the patient has had a liter of anti-pneumococcus serum without apparent benefit, further use of serum seems unwise The heart examination reveals no evidence of endocarditis

January 7th Blood-culture, pneumococcus Type I

Heart sounds fair quality a soft systolic murmur at apex is heard

January 8th The patient visited at 9 45 A M Was quiet, apparently sleeping When seen two hours later the breathing stertorous, ptosis of the left eyelid, right side of face smooth, with the left corner of the mouth drawn to the left, and great weakness of the right arm and leg This condition persisted, and patient became more and more stuporous, pulse and temperature rising, dying in the early morning of January 10th

When the serum failed to clear the blood of pneumococci, some complication was immediately suspected The particular type of complication most likely to cause a bacteriemia to persist is, of course, an endocarditis, and this was suspected before any change in the physical signs of the heart occurred Other complications were, of course, sought There was no evidence of abscess, empyema, meningitis, or pericarditis On the 7th of January a systolic murmur appeared, but while this is very suggestive, it is not conclusive, for patients very ill for days may develop a functional cardiac murmur On January 8th, however, additional evidence appeared in the sudden appearance of a hemiplegia Here again this could be due to cerebral hemorrhage, but with the persistence of the bacteriemia and the development of the cardiac murmur we were strongly inclined to believe this phenomena to be due to an embolus Evidence of other emboli was sought for, but not found

Autopsy Lobar pneumonia involving the entire right upper and posterior portions of lobe, posterior half of left upper, and entire left lower lobe, acute fibrinous pleurisy, most marked at the right base, abscess in apex of right lung (a cavity measuring about 3 cm in diameter), acute splenic tumor, small infarcts in spleen, parenchymatous degeneration of the liver and kidneys, chronic pancreatitis, fat necrosis in the pancreas, and acute vegetative endocarditis of the mitral valve (pneumococcus)

Heart weighs 350 grams

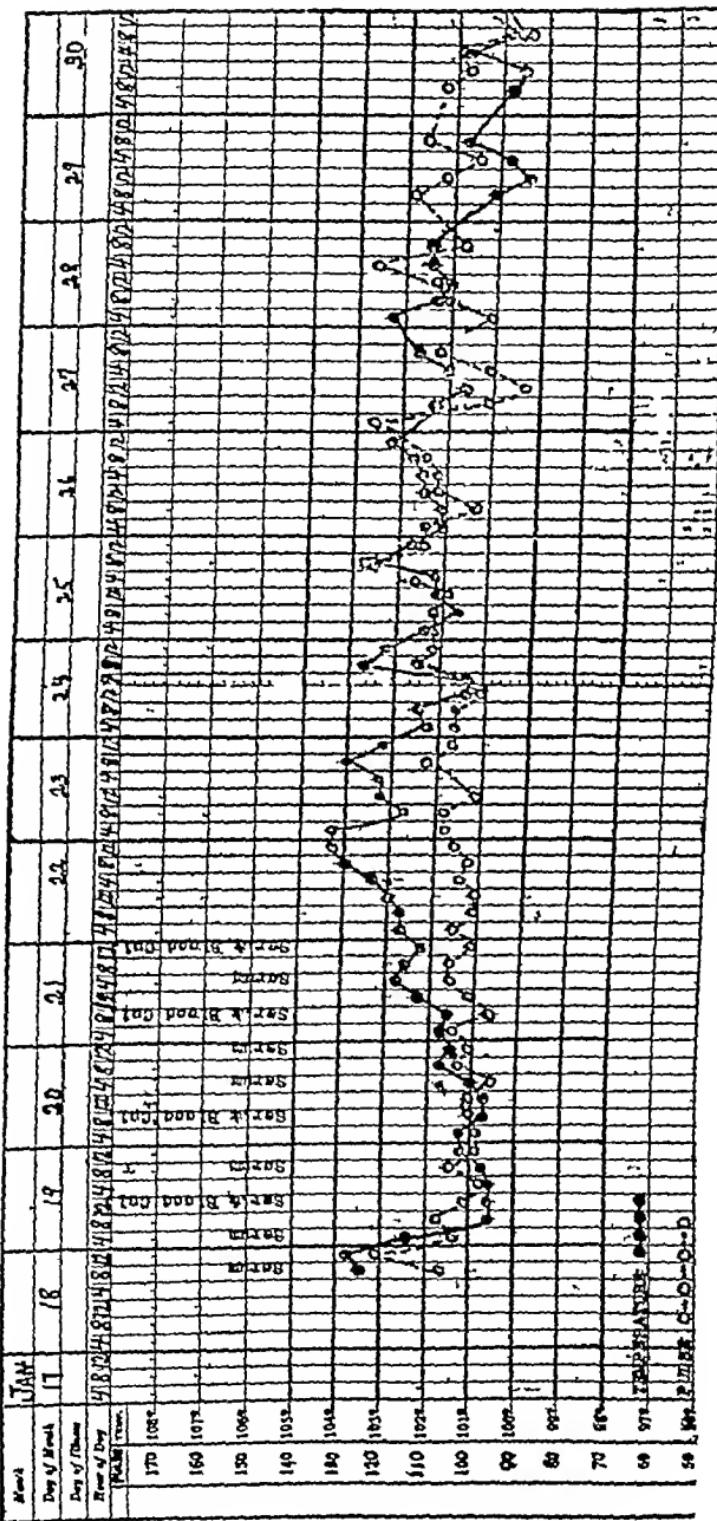
MEASUREMENTS

Pulmonary valve	Normal.
Tricuspid valve	Admits three fingers.
Mitral valve	Admits two fingers.
Aortic valve	Normal.
Thickness of right ventricle	0.3 cm. in thickness.
Thickness of left ventricle	1.6 cm. in thickness.

The pericardial surface is smooth and shining, pericardial fat slight in amount. All chambers of the heart are filled with post-mortem clot, no thrombi present. All of the valve cusps are normal except the mitral. On the auricular surface of both mitral leaflets there are four vegetations about equal distance apart, attached about the line of closure. Two of the vegetations are rather large, measuring approximately 0.5 cm in height and perhaps 2 mm in width. They are opaque, grayish-white in color, and look firm, as though organized. About their bases they can, however, be crushed readily, and obviously are recent in origin and composed almost entirely of fibrin and platelets. There is no evidence of an old valvular lesion. There are no vegetations on the auricular wall or on the chordæ anywhere. The cardiac musculature is pale, but otherwise normal.

Smears taken from the abscess in the lung and the heart vegetations revealed Gram positive, lancet shaped diplococci with capsules present—large numbers. Unfortunately, the cultures failed to grow.

To make this case complete, a head examination should



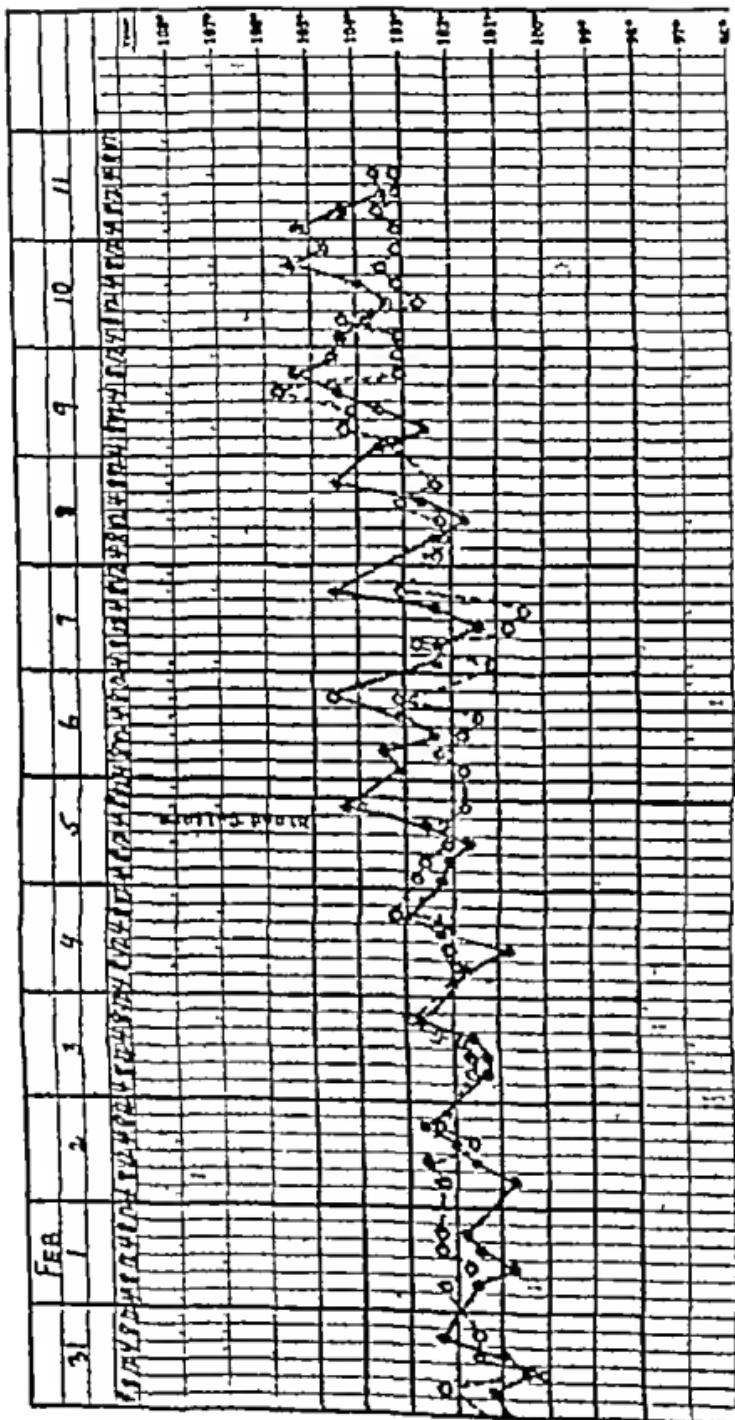


Fig. 196.—Chart of Case III (continued)

have been made, and it is to be regretted that this was not allowed

You will undoubtedly ask the question, Did not the abscess play a part in the persistence of the bacteremia? It is impossible to say dogmatically that it did not, but it is a matter of common clinical knowledge that abscesses following pneumonia frequently occur without an accompanying bacteremia

CASE III (Chart III, Hospital No 37,109) —A negress, aged thirty-five, laundress, born in Georgia, entered the hospital for the first time January 17, 1918, complaining of weakness and a "bad cold" of ten days' duration. She has been married for five years, has one child four years old and well. One child died at four months of pneumonia, and two miscarriages "one week before term." She has always considered herself well and strong. Five years ago she had pneumonia associated with jaundice. One year ago, sore on vulva followed by rash (Figs. 195, 196)

Ten days before entrance the patient got her feet wet. The next morning she was suddenly seized with violent headache, which very shortly was followed by a severe chill, which in turn was followed by fever and prostration. A day later the headache disappeared and there appeared in her left side a severe knife-like pain, which was made worse by deep breathing and cough. She has been much prostrated with the fever, pain in the side, and cough since onset. The day before she entered the hospital she noticed that her fingers were yellow and that her urine was a very light yellowish color.

The patient is a well-developed, well-nourished colored woman, acutely ill, breathing rapidly, apparently in considerable discomfort. Frequent cough. Scleræ are yellow. Over the left lower lobe posteriorly there is dulness, bronchial voice, and breath sounds with many crepitant râles.

Examination of the heart. The apex-beat is not seen nor felt. No thrills. The area of cardiac dulness is only slightly increased. The sounds are of good quality and no murmurs are heard. The aortic second is not accentuated.

The diagnosis of the acute condition in this case, of course,

presents very little difficulty. Both history and physical examination are typical of lobar pneumonia. The history of miscarriage and sore on vulva would lead us to suspect syphilis. As we stated at the outset, we shall spend little time on the disease pneumonia, but dwell rather on the special features of the case in connection with the heart.

January 18th		W B C., 25,000 88 per cent polynuclears. Urine orange colored 1025 acid albumin a heavy trace. Few granular casts bile present. Sputum, pneumococcus, Type I Blood-culture, pneumococcus, Type I Wassermann alcoholic antigen +++++ cholesterol +++++
	4.00 P. M.	1/2 c.c. of Wassermann injected subcutaneously was followed by no reaction.
	8.00 P. M.	100 c.c. antipneumococcus, Type I intravenously
January 19th	12.00 M.	Blood-culture, Type I pneumococcus. 100 c.c. antipneumococcus serum Type I intra venously
	8.00 P. M.	100 c.c. antipneumococcus serum Type I intra venously
January 20th	9.00 A. M.	Blood-culture, Type I pneumococcus.
	9.00 A. M.	100 c.c. antipneumococcus, Type I intravenously
	4.00 P. M.	100 c.c. antipneumococcus, Type I iotrauenously
	12.00 P. M.	100 c.c. antipneumococcus, Type I intravenously Note on this day is that patient is much more comfortable and jaundice much less marked.
January 21st		W B C., 36,000
	9.00 A. M.	Blood-culture Type I pneumococcus. 100 c.c. antipneumococcus serum Type I intra venously
	4.00 P. M.	100 c.c. antipneumococcus serum, Type I intra venously
	12.00 P. M.	100 c.c. antipneumococcus serum Type I intra venously

Blood-culture taken just before the midnight dose of serum showed Type I pneumococcus.

For the first time, on January 21st, a systolic murmur at the apex was noted. The lung signs up to this time revealed no evidence of any spread of the process, nor was there any reason to think the affected lobe was breaking down with abscess formation. There were no signs of meningeal involvement.

Up to this point the striking feature of the case was the fact that after the first dose of the serum the blood still contained the type of pneumococcus which in the light of Case II, particularly without other obvious complications, suggests immediately a pneumococcus endocarditis. No cardiac murmur could be made out at this time. Subsequent serum treatments failed to clear the blood of this type of pneumococci. The patient had in all 900 c.c. of antipneumococcus serum. This is a very large amount when we consider that in the successfully serum treated cases of Type I infections require on the average of only 200 to 300 c.c. of serum. With the development of the murmur on January 21st our suspicion of an endocarditis received very convincing support. Further events bear out our early ideas of the situation.

January 25th Signs of resolution (many moist rales, decrease in dullness and bronchial character of the voice and respiration) are present at the extreme base of the lung. Apex murmur remains about the same as on January 21st.

January 28th Blood-culture Pneumococcus, Type I, present

January 31st Lung signs rapidly clearing up

February 4th Roentgenograms reveal no disease of the lungs.

February 5th The systolic murmur at the apex is louder and harsher than when last noted. Just along the left sternal margin in the second and third interspaces is heard a short soft diastolic murmur. A capillary pulse is present in lips and fingers. Blood-culture Pneumococcus, Type I, average number of colonies per cubic centimeter, 31.

Note—Patient's family suddenly decided to take the patient home on February 11th. The visiting nurse reported that the patient died four days after leaving the hospital.

While we have no autopsy in this case, the clinical evidence is very conclusive that we are dealing with a case of acute pneumococcus endocarditis developing during the course of acute lobar pneumonia. In this case there is the presence of syphilis, which may have been a predisposing factor. We know that acute endocarditis of this type is more prone to develop on valves which have been previously diseased, as in old rheumatic endocarditis and syphilitic endocarditis. No

evidence that there was an old syphilitic endocarditis is present. That is a possibility, however, but not a very great one.

In conclusion, we may call attention to the fact that acute pneumococcus endocarditis may be diagnosed with some certainty in the Type I pneumococcus pneumonia before cardiac signs appear. This is of importance from a prognostic standpoint.

CLINIC OF DR T STUART HART

PRESBYTERIAN HOSPITAL

MITRAL STENOSIS AND AURICULAR FIBRILLATION DIGITALIS—ITS USES AND DANGERS

THIS afternoon, gentlemen, I wish to discuss with you not a rare, not a spectacular condition, but an exceedingly common one, a condition which each one of you will meet frequently in your practice, namely, *mitral stenosis* associated with *auricular fibrillation*. Although these cases are occasionally puzzling, their diagnosis usually presents but little difficulty. The treatment of many of them is most satisfactory, but in order to secure satisfactory results each patient must be individually considered, they need constant supervision, and the details of their treatment must be modified from time to time as the special indications develop. It is this question of individualization to which I wish to especially invite your attention.

CASE I.—The first patient which I wish to present for your consideration is a man sixty-one years of age whom I have asked to give up his work for the afternoon and to come before you because he represents in a most interesting manner a sequence of events to which I wish to direct your attention.

As a child he had a severe attack of scarlet fever. Following this he had several attacks of tonsillitis, and at nineteen years of age a Neisserian infection. With the exception of these illnesses he was perfectly well. He has never had acute rheumatic fever. He denies luetic infection. He has used alcohol moderately, never to excess. He has never used tobacco. When twenty-three years of age, while occupying a clerical position which he has continued ever since, it was accidentally discovered that he had a heart murmur. At this time he had no subjective symp-

toms, but was told that he had a valvular lesion and was advised to avoid excessive physical exertion. He has accordingly always led a quiet life, keeping in mind the possible danger from his valvular defect and avoiding any considerable bodily activity. The first subjective symptoms which appeared were twenty years later when he was forty-three years of age. Up to this time he had been perfectly well and had no symptoms referable to his heart. He then began to notice that occasionally he had a sensation of "thumping" in the precordial region, and discovered that his heart was irregular. Beyond this, however, he suffered no inconvenience, but continued his quiet mode of life, at this time attributing these occasional attacks of irregularity to gastro-intestinal disturbances.

He first came under my observation in 1909 when he was fifty-one years of age. At that time there was nothing in his physical examination that was abnormal except the condition of the heart, which showed the following features. The apex was palpable in the fourth space 9 cm to the left of the midsternal line, dulness 10 cm to the left of the midsternal line, none to the right of the sternum. No thrills could be detected. The first sound at the apex was snappy and was preceded by a rough crescendo murmur, terminating abruptly in the first sound. Along the left edge of the sternum with maximum intensity in the third space was heard a loud diastolic murmur. The sounds at the base of the heart were normal. During the period of the examination occasional extrasystoles were detected. At this time there was no evidence of cardiac insufficiency. In January, 1911, he developed an attack of bronchopneumonia. At the onset of this attack the heart showed no changes from the condition which I have described to you, but during the course of this disease the heart became suddenly very irregular and increased somewhat in size, but not to any considerable degree. He was given digitalis. The heart rate was gradually controlled and he made a satisfactory recovery from his pneumonia. He was discharged with the advice to limit his physical activities and to continue taking digitalis. The latter, however, he neglected to do, and in the latter part of April of this same year he

presented himself complaining of a moderate degree of dyspnea on slight exertion and slight pretibial edema. His heart showed complete irregularity, a rate counted at the apex of 132 and a radial pulse of 104. The heart apex was farther down and more to the left than on former examinations. It was located in the fifth space 11 cm to the left of the midsternal line. He was at once put to bed and given effective doses of digitalis. In two weeks the heart rate was reduced to 81 per minute, the radial rate was 74 per minute. He was then allowed to leave his bed and to gradually resume his office work. His greatest desire at that time was to be able to continue his work, upon which the support of his family depended. He was, therefore, instructed to save his physical exertion in every possible way. He accomplished this by never climbing stairs, by using the surface cars rather than the subway or elevated, by returning home as soon as his work was completed and going immediately to bed, staying there until it was time for him to get up and return to his office the next morning. After a time these restrictions were made less rigid, and at the present time he is able to lead a fairly normal life as to hours, but his physical activities are always limited. During this whole period he has never failed to take his daily dose of digitalis in amounts which I have found were suited to his individual necessities. At the present time his heart shows complete irregularity. The apex is in the fifth space 13 cm to the left of the midsternal line. The presystolic and the diastolic murmurs are still present and, in addition, there has developed a loud systolic murmur heard with maximum intensity at the apex. The lungs are clear. The liver is palpable 2 cm below the costal margin. There is no edema of the extremities. His color is good. There is no dyspnea on the exertion to which he is accustomed.

It is evident that we are dealing with a case of chronic cardiac valvular disease. Let us briefly review the history together, considering *first* the cause of the valvular defect, *second*, the anatomic condition, and *third*, the changes in the functional activity of the heart. In his story we have an account of three infections—scarlet fever, repeated tonsillitis, and gonorrhea.

It is impossible to say which one of these infections was the cause of the cardiac condition, but it is most probable that the scarlet fever afforded the infection which began to deform the heart valves

It is clear from the evidence that his early valvular defect was a mitral stenosis. The mitral valve evidently became more and more deformed until at the present time it is clear that he has a mitral insufficiency as well as a stenosis. Hand in hand with the changes in the valves has developed a change in the heart muscle. At first there was little or no change in the size of the heart. At the present time there is evidence of a considerable hypertrophy of the left ventricle. This, of course, is nature's method of adjusting the heart to meet the conditions arising from the valvular defect. The ventricle is thus strengthened and is better able to maintain the circulation.

The first symptom of which this patient was conscious was a "thumping of the heart," and this, he himself discovered, was due to irregularity in its action. As this irregularity was present when he first came under my observation I can describe to you its features as I found them when I made the examination. When I felt the pulse at this time I found that for considerable periods it was perfectly rhythmic, with good sized pulse waves following each other in normal succession, then there would appear a small wave which occurred a little too early and which was barely palpable. This was followed by a pause which was longer than the interval between two of the rhythmic pulse waves. The pause, in turn, was followed by a large wave, and then there was a succession of regular rhythmic pulse waves. When one auscultated the heart a similar sequence of events was evident. For the most part the heart was perfectly rhythmic, but occasionally there was a beat which occurred a little too early, this was followed by a pause and then the rhythmic series was continued. You will thus recognize this as a description of an extrasystole, as you know the extrasystole is due to a contraction of the heart the stimulus for which arises from some portion of the heart muscle other than the sinus node where the impulses normally originate. Some portion of the heart muscle becomes

irritable and initiates a contraction before the time that the normal contraction which arises at the sinus node appears. The next impulse coming from the sinus node finds the heart muscle exhausted and unable to respond to this stimulus, that is, the heart muscle is in its "refractory period," hence it does not contract, and there is a pause which permits it to recover, and it is ready to respond to the next physiologic stimulus coming down from the sinus node.

The sensation of "thumping" is a common description given by the patient to this form of irregularity. His sensation is due to the fact that the contraction does not pass over the heart muscle by the normal path, and therefore is ineffectual in emptying the ventricles, giving rise to this abnormal sensation in the chest. You will notice that notwithstanding the easily recognized valvular defect, the change in the size of the heart and the presence of extrasystoles, that this patient developed no symptoms of cardiac insufficiency for a period of over thirty years.

Eight years ago he had an attack of pneumonia. During this time the heart became completely irregular and symptoms of insufficiency first developed. By complete irregularity we mean a heart activity in which we cannot predict what the next event is to be. Heart beats follow each other in irregular succession without rhyme or reason. We have a series of contractions following each other in rapid succession, and these are separated by long or short intervals. The time relations and volume of the successive waves of the pulse are correspondingly irregular, and we have a haphazard succession of large and small waves separated by longer or shorter intervals which occur in utter confusion. There are several conditions which may give rise to complete irregularity of the heart action. Only one or two of these, however, need arrest our attention. By far the most frequent cause of complete irregularity is the condition known as (1) fibrillation of the auricles. Other conditions which one should bear in mind are (2) a sinus arrhythmia in which the normal pace-maker at the sinus node loses its usual rhythmicity, but still retains its function as pace maker of the heart, and (3) the very frequent occurrence of extrasystoles at irregular intervals so dis-

torting the normal rhythm that it gives one the impression of complete irregularity. Auricular fibrillation, however, is by far the most frequent cause of complete irregularity, and it is the cause of this type of activity in at least 95 cases out of every 100. We may obtain corroboration of this condition by inspecting the veins of the neck when their pulsation is prominent enough to give us satisfactory information. When the auricles are fibrillating there is no presystolic wave to be detected and all the venous waves occupy a time synchronous with ventricular activity. Another point which may help us to differentiate the development of auricular fibrillation is the fact that the presystolic murmur which has been present usually disappears and is

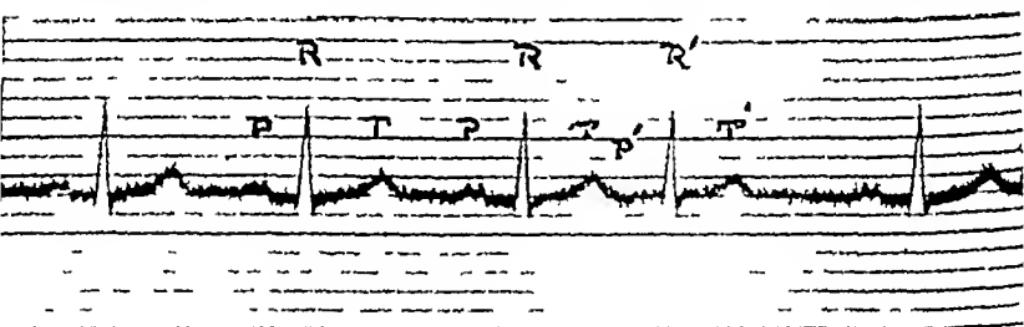


Fig 197—June 7, 1910. Auricular extrasystole (P' R' T) with compensatory pause. Note notch in P (auricular wave), indicating a defect in the auricular muscle.

replaced by a diastolic murmur terminating a short time before the first sound. That this is not, however, always a constant feature is illustrated by the present patient, who with fibrillating auricles still retains a definite crescendo presystolic murmur terminating abruptly in the first sound.

As you know, the most accurate way of obtaining information in regard to the arrhythmias of the heart is by means of the electrocardiogram, and I therefore wish to direct your attention to the records of this patient.

His first record (Fig 197) was taken in June, 1910. You will notice that for the most part the rhythm was perfectly regular, but at one point an extrasystole appears which is a contraction

occurring a little too early and is followed by the so-called compensatory pause. You will notice that the ventricular portion of the extrasystolic complex is identical in form with those of the rhythmic contractions, but that the interval between the wave which precedes this complex and the extrasystole is so short that the P wave (auricular complex) is superimposed upon the preceding T wave. This is an auricular extrasystole, which is to say that a point in the auricular wall has become overirritable and initiates this contraction. There is one other point of interest in this record to which I wish to call your attention, and that is, that the P wave of all the complexes is slightly notched. This is a deviation from the normal and indicates that the impulse is

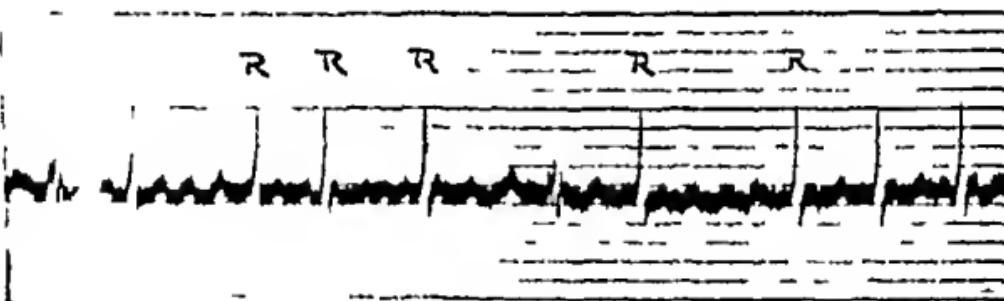


Fig. 198.—April 28, 1911. Auricular fibrillation. Rapid and very irregular. Note absence of P waves and presence of small irregular oscillations due to the fibrillating auricles. At this time the patient showed signs of cardiac insufficiency.

passing over the auricle by an abnormal path, in other words, it shows us that the auricular muscle is already slightly damaged.

The next record (Fig. 198) was taken in April, 1911. This, you will recall, was several months after his attack of pneumonia, during which he began to have complete irregularity. It was at this time that he was showing signs of cardiac insufficiency. As you will recognize, the record is one typical of auricular fibrillation. The rate is rapid, there is complete irregularity, the P wave is absent, and throughout the whole record appear small undulations characteristic of a fibrillating auricle.

Figure 199 is a portion of his electrocardiogram which was taken in April, 1915. At this time the heart was much slower

and compensation was well established. You will notice that is much slower, the rate being 68 per minute. This condition was brought about by an improvement in the heart muscle, an important element in the heart control being the continuous administration of digitalis.

The next point to which I wish to call your attention is the value of the pulse deficit as a clinical guide to our dosage of digitalis. In cases of auricular fibrillation with a rapid rate and a very irregular heart action frequently the number of the impulses which can be counted in the radial pulse is far below the actual number of cardiac contractions. Only those waves which are of considerable volume and force can be felt at the wrist, and

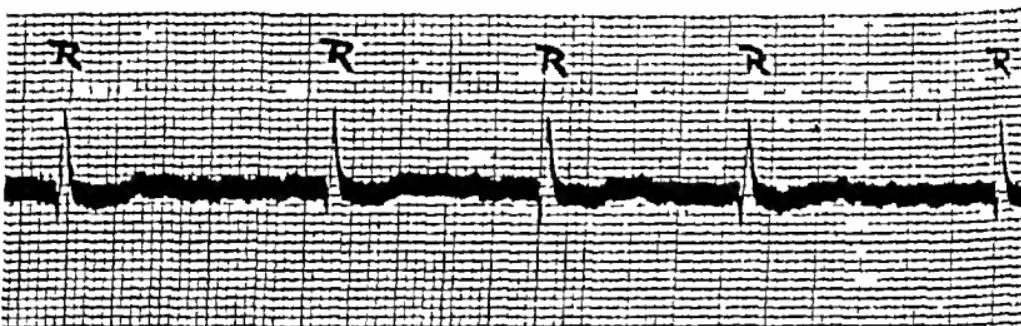


Fig 199—April 9, 1915. Auricular fibrillation under suitable dosage of digitalis. Heart action slow and efficient.

all the evidence of auricular fibrillation persists, but that the heart many small ventricular contractions expend their force before reaching the radial artery, and may even fail to open the aortic valves. These small contractions are ineffectual in maintaining an adequate circulation, yet are exhausting to the heart muscle, for we know that in accordance with the law discovered by Bowditch, every contraction of the heart muscle is maximal, that is to say, if it contracts at all it exhausts all of the energy stored as contractile material in its muscle-fibers at any particular moment, hence it is evident that however small the contraction may be, it must be taken into consideration in estimating the gravity of the condition of any particular heart. The inadequacy of the observations on the radial pulse alone is well illustrated in Fig 201.

Here the lower margin of the shaded area indicates the radial count. If one were guided by this alone one would have said that on admission the cardiac rate was under 100 and usually about 70. The upper boundary of the shaded area is the count taken by auscultation at the apex, and represents much more accurately the true condition, the admission rate being 130, the gradual reduction to the neighborhood of 60 making the improvement apparent. The term "pulse deficit" is used to designate the difference in the count when taken at the apex by auscultation and at the radial pulse by palpation. Under rest and digitalis the general circulatory conditions usually improve progressively, the heart rate slows, and the pulse deficit disappears or

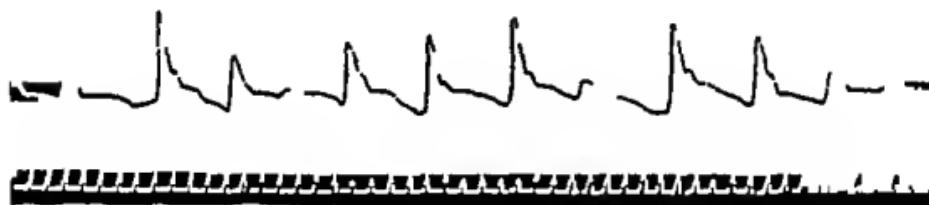


Fig 200.—Radial pulse tracing

becomes insignificant. I am sure that in following your cases you will find it of great advantage to keep a graphic chart of this kind, as it is an important aid in indicating the progress of the case and of regulating the digitalis administration.

Another point to which I wish to direct your attention is the question of blood pressure in auricular fibrillation. Figure 201 is a tracing of the radial pulse of our patient at the time that he was showing cardiac insufficiency. Merely a glance at this record is sufficient to indicate to you that each individual pulse wave has a different pressure value. The blood pressure obtained by the ordinary methods gives a very inaccurate idea of the actual condition. A much more accurate method is to determine the "average systolic blood pressure" by the "fractional method," which is secured in the following way:

The apex and radial are counted for one minute, then a blood pressure cuff is applied to the arm, and the pressure raised until the radial pulse is completely obliterated, the pressure is then lowered 10 mm and held at this point for one minute, while the radial pulse is counted, the pressure is again lowered 10 mm and a second radial count is made, this count is repeated at intervals of 10 mm lowered pressure until the cuff-pressure is insufficient to cut off any of the radial waves (between each estimation the pressure on the arm should be lowered to 0) From the figures thus obtained the average systolic blood-pressure is calculated by multiplying the number of radial beats by the pressure under which they came through, adding together these products and dividing their sum by the number of apex-beats per minute, the resulting figure is what we have called the "average systolic blood-pressure" The following two observations made on this patient will indicate the method of computation

April 29, 1910 Apex, 131, radial, 101, deficit, 30

Brachial pressure	Radial count
100 mm	0
90 mm	13 $13 \times 90 = 1170$
80 mm	47 - 13 = 34 x 80 = 2720
70 mm	75 - 47 = 28 x 70 = 1960
60 mm	82 - 75 = 7 x 60 = 420
50 mm	101 - 82 = 19 x 50 = 950
	Apex = 131) <u>7220</u>

Average systolic blood pressure, 55+

May 11, 1910 Apex, 79, radial, 72, deficit, 7

Brachial pressure	Radial count
120 mm	0
110 mm	44 $44 \times 110 = 4840$
100 mm	64 - 44 = 20 x 100 = 2000
90 mm	72 - 64 = 8 x 90 = 720
	Apex = 79) <u>7560</u>

Average systolic blood pressure, 95+

The blood-pressure determinations made in this way are of practical value in grouping our cases and in following their progress In general, I may point out that fibrillating auricles occur mainly in two pathologic conditions (1) cases of mitral

stenosis following the acute infections, and (2) cases of general arteriosclerosis often without valvular defects. The patients be-

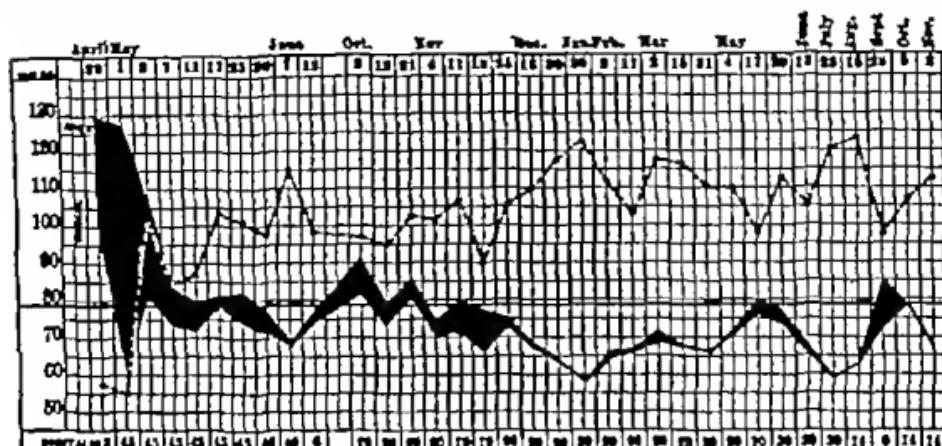


Fig. 201.—The shaded area represents the pulse deficit the upper edge is the apex rate the lower edge the radial rate. The unbroken line is the average systolic blood pressure. The figures in the digitalis column indicate minims of the tincture per day. Patient not confined to bed except for first fourteen days.

longing to Class I show a very low average systolic blood pressure during the stage of cardiac insufficiency (Figs. 201 and 202), as

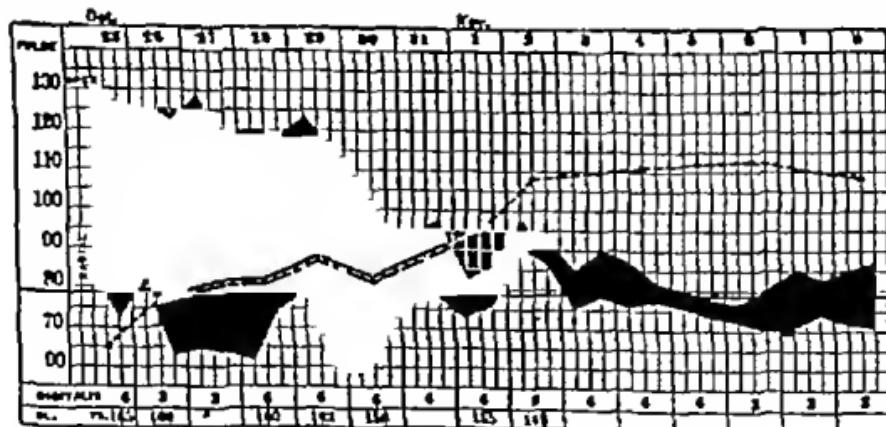


Fig. 202.—Another case showing pulse deficit and increase in average systolic blood pressure under rest and digitalis. The lower figures indicate blood-pressure estimations taken in ordinary way these are very misleading

the force of the heart improves the pressure gradually rises, this is a good prognostic sign. The patients belonging to Class II

show a blood-pressure nearer the normal, and with improvement the pressure usually increases, and ultimately they show a condition of hypertension

The treatment of this patient may be summed up in two words —rest and digitalis. As long as the patient shows cardiac insufficiency, a rapid heart, or any considerable pulse deficit, he should be kept in bed, and this should be continued long enough after the heart has become slow to allow an opportunity for hypertrophy, which progresses most satisfactorily when the diastolic rest of the heart is reasonably prolonged. This is because the blood-supply of the heart is best during the diastolic period, and if this period is too short the heart muscle does not receive an adequate supply of nutriment.

Digitalis is almost a specific in these cases. As you know, digitalis has the property of depressing conductivity. In the type of cases which we are considering the pathologic process in the heart muscle is of such a nature that it renders it particularly susceptible to the action of digitalis. Experimental investigations have shown that the depression of conductivity produced by digitalis is accomplished in two ways: first, by stimulation of the vagus, and second, by a direct action on the muscle-fiber of the heart. The effect of the administration of digitalis is to make the passage of stimuli from the auricles to the ventricles through the bundle of His more difficult. As digitalis becomes effective, more and more of the haphazard rapid and irregular impulses from the auricles are blocked. As a consequence the ventricles receive fewer stimuli and become slower and contract at more uniform intervals. They never, however, become perfectly rhythmic in their contractions.

What preparation of digitalis shall we select to obtain the most satisfactory results? I think it makes very little difference as long as you use a preparation which is active and with which you are familiar. I wish to emphasize the fact that it is most important for you to keep thoroughly familiar with the preparation which you use, you will then know whether you are giving doses which are adequate to produce the results for which you are looking. For administration by mouth I know of nothing

better than a good tincture which has been properly standardized and which is known to be active. I never use digitalis subcutaneously. I have yet to find a preparation of digitalis which is active which does not produce a very painful reaction at the point of injection. If digitalis is indicated, as is sometimes the case, in massive doses rapidly administered, the only satisfactory way to do is to use it intravenously. For this purpose digi puratum and digifolin are excellent preparations. This method should never be employed if a patient has been previously taking digitalis, since it is then impossible to estimate how much of the drug the individual will tolerate. In cases with rapid heart, a considerable pulse deficit, low blood-pressure, dyspnea, edema, and other signs of cardiac insufficiency one may begin by giving of the tincture 60 to 90 minimis in the first twenty four hours, or digifolin intravenously, 1 c.c. every six hours. After the first twenty four hours the effect of the administration should be carefully watched, and as the heart responds the amount of the drug should be gradually diminished.

The rule for giving digitalis in these cases is to give it until you get the physiologic effect. The heart must be brought under the effect of digitalis and must be kept digitalized. The best results are obtained when one keeps the heart rate between 60 and 70, with no deficit. It is usually necessary for these patients to continue digitalis throughout their lifetime. There are occasional cases who improve to the point where they can get along without the drug, but these are very few and far between. I want to urge you most emphatically to give digitalis in these cases continuously. I see more errors committed through the giving up of digitalis in these cases than by using too much of it.

Recapitulation—This patient illustrates

- (1) That for many years a marked valvular defect may cause no cardiac insufficiency
- (2) The early evidence of auricular myocardial change, viz., auricular extrasystoles and changes in the auricular complex of the electrocardiogram
- (3) The transition to auricular fibrillation under the stress of a new infection.

(4) The development of cardiac insufficiency when the haphazard impulses from the auricles produced a rapid, irregular, and ineffectual activity of the ventricles, with marked pulse deficit and a low blood-pressure

(5) The general circulatory improvement associated with slowing of the heart, loss of pulse deficit, and increase in the blood-pressure under rest and digitalis

(6) The maintenance of a fairly satisfactory circulation for many years by moderately restricting his physical and emotional activities and by the judicious and continuous use of digitalis

CASE II.—In contrast to the first patient whom I presented to you this afternoon, I will ask you to examine with me this young woman, thirty-five years of age, single, and a stenographer. She first came to the hospital on September 19, 1917, complaining of weakness, exhaustion, digestive disturbance, and shortness of breath. As a child she had measles, whooping-cough, and diphtheria. After this she was perfectly well until at the age of twenty-four she had a severe attack of acute rheumatic fever of which the outstanding feature was a multiple arthritis of considerable severity with fever which confined her to bed for a period of six weeks. She tells me that a murmur was discovered in her heart three years after her attack of rheumatism, that is, seven years ago. She, however, noticed no subjective discomfort from this until four years ago, when she began to be short of breath on climbing stairs and developed a cough accompanied by expectoration of mucus streaked with blood. Soon after this she began to suffer from digestive disturbances. These consisted of pain in the epigastrium after eating, nausea, the formation of a great deal of gas, and from time to time attacks of vomiting.

One night in May, 1917, she suddenly was conscious of severe palpitation of the heart. This symptom had never been present before, but has continued ever since. The day following the onset of the palpitation she had a very severe gastro-intestinal upset, with vomiting and retching. She was at that time working in South America, and went to a hospital there and stayed there for three months, gradually improving

until she was able to walk about and felt reasonably well. She then came home, but was very seasick during the voyage, and on reaching New York was very short of breath and unable to walk more than a few steps. Some weeks after this she was admitted to the Presbyterian Hospital. At that time she was dyspneic and orthopneic. The veins of the neck were full and showed a marked systolic pulsation. The lungs showed dulness and many fine, moist rales at both bases. The heart was enlarged both to the right and to the left. In the apical region could be felt a presystolic thrill, with diastolic and systolic murmurs loudly heard. The pulmonic second sound was loud and accentuated. The heart was completely irregular, apex rate 156, radial 88, deficit 68. The liver could be felt in the right midclavicular line 3 cm below the costal margin. The spleen was easily felt and was quite tender. There was no edema of the extremities. The diagnosis was made of chronic cardiac valvular disease of rheumatic origin, mitral stenosis and insufficiency, cardiac dilatation, auricular fibrillation, cardiac insufficiency. She was kept absolutely at rest and was given 45 minimis of tincture of digitalis a day. Six days after admission the apex rate was 80, radial 60, deficit 20, and she was feeling much more comfortable. She was discharged considerably improved on October 18th (apex rate 64, radial 60, deficit 4), with instructions to limit her activities and to continue her digitalis, 10 minimis of the tincture twice a day. However, she thought this was unnecessary and discontinued its use, and in spite of making very little physical effort, her heart again soon became insufficient and she returned to the hospital on October 30th in much the same condition in which she had been admitted in the middle of September. From that time up to the present she has spent at least two-thirds of her time in the hospital, improving while she is at absolute rest, but soon breaking down when she attempts to live at home.

This woman presents a very different picture from that exhibited by our first case. Instead of walking, she comes here on a stretcher. She is not, however, lying down flat, but is propped up in a sitting posture, since it is impossible for her to

breathe with comfort when her head is lower than at present. Even reclining quietly she is evidently dyspneic. Her face is worn and shows her evident anxiety. The skin and mucous membranes are pale. There is no cyanosis. The examination of the lungs shows dulness and many fine, moist râles at both bases. Over the lower portion of the back there is some subcutaneous edema which pits on pressure. The liver percusses from the fourth space and its edge is felt 5 cm below the costal margin and is pulsating. The spleen is easily felt and is quite tender. There is no free fluid in the abdominal cavity. The legs show slight pretibial edema. I have outlined the borders of the heart as they are made out by percussion with a heavy black mark. You will observe that these are enlarged, extending in the sixth space to the anterior axillary line on the left and 5 cm to the right of the midsternal line in the second and third spaces. There is, however, no bulging of the precordial region, but a considerable outward heave of the chest with each contraction of the heart. On palpation one can feel the pressure produced by the cardiac thrust, and in the region of the apex there is felt a fine thrill which is diastolic in time. On auscultation there is heard at the apex a long, harsh diastolic murmur, a loud broad first sound, a systolic murmur, and a sharp second sound. At the base the pulmonic second sound is accentuated and greater than the aortic second in intensity.

On account of the exquisite tenderness in the apical region I will not ask you to make a complete examination of the heart, but I want a number of you to feel the radial pulse, to observe the pulsations of the veins of the neck, and to listen to the heart in the precordial region near the base where you can study the rhythm without discomfort to the patient. On examining the pulse you will notice that it is very slow and in almost perfect rhythm at a rate of 42 per minute. The volume is fairly good, there is no evidence of arterial thickening. On listening to the heart one finds, first of all, that the rate is 84 per minute, exactly twice the rate as determined in the radial pulse. The heart sounds also show a very striking rhythm. There is a strong beat followed shortly by a weaker beat, which is, in turn,

followed by a long pause, again a strong beat, an early weak beat, and a long pause. If, while you are listening to the heart, you observe the veins of the neck, you will notice that they are quite full and pulsating clearly. There is a pulsation in the veins corresponding and synchronous with each heartbeat and the rhythm is identical with that of the heart, viz., a large wave, an early small wave followed by a long pause. There is no evidence in the veins of the neck of a presystolic wave which should be present under normal circumstances. This rhythm which you have observed is known as the "coupled rhythm."

As this sort of examination is exciting to the patient, we will now let her return to the ward and will discuss some features of her condition as you have seen them after she has left us. She presents a typical picture of mitral stenosis and insufficiency with cardiac dilatation and marked evidences of cardiac insufficiency. This, of course, has followed her severe attack of rheumatic fever, and it is clear from her history that auricular fibrillation suddenly commenced in May, 1917.

When this patient was first seen she was put on digitalis and responded to its use in a very satisfactory manner.

The heart became slow, the pulse deficit disappeared, the general circulation greatly improved, and she was able to leave the hospital in fair condition. Her electrocardiogram (Fig. 203) taken at this time shows a considerable control of the heart rate.

Since that time, however, on each occasion that she has been in the hospital it has been more and more difficult to improve the functional efficiency of the heart. The difficulty arises from the fact that the myocardial lesion has been progressive and the contractile power of the ventricular muscle has progressively diminished with an evident loss of muscle tone. Hand in hand with these changes there has appeared an increased susceptibility to digitalis, until at the present time it is almost impossible to administer doses which effectually control the heart rate without eliciting toxic symptoms.

We know experimentally that the fundamental properties

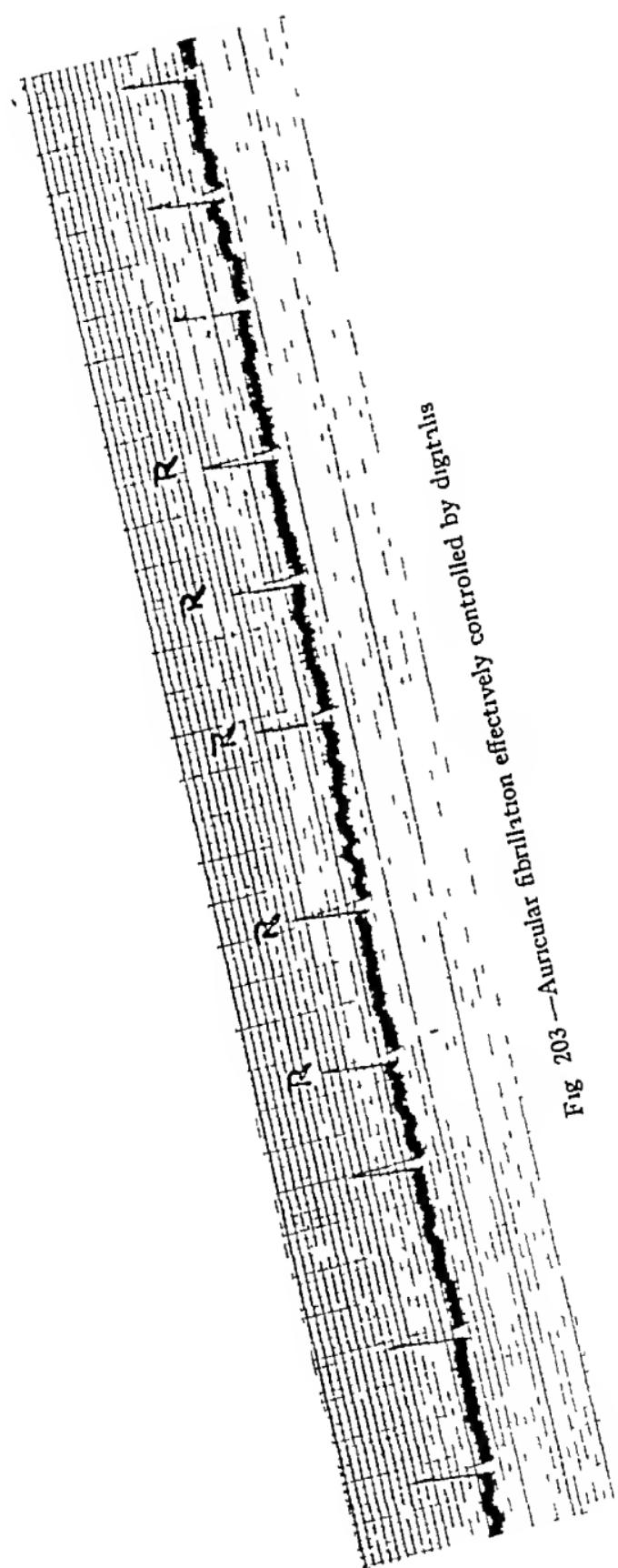


Fig. 203.—Auricular fibrillation effectively controlled by digitalis

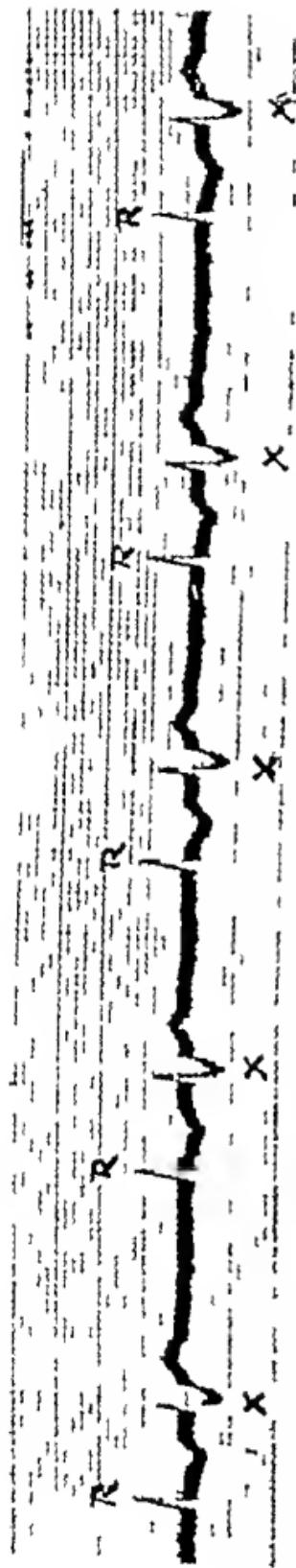


Fig. 204.—Auricular fibrillation too much digitalis. Complete heart block and ventricular extrasystoles producing the coupled rhythm.

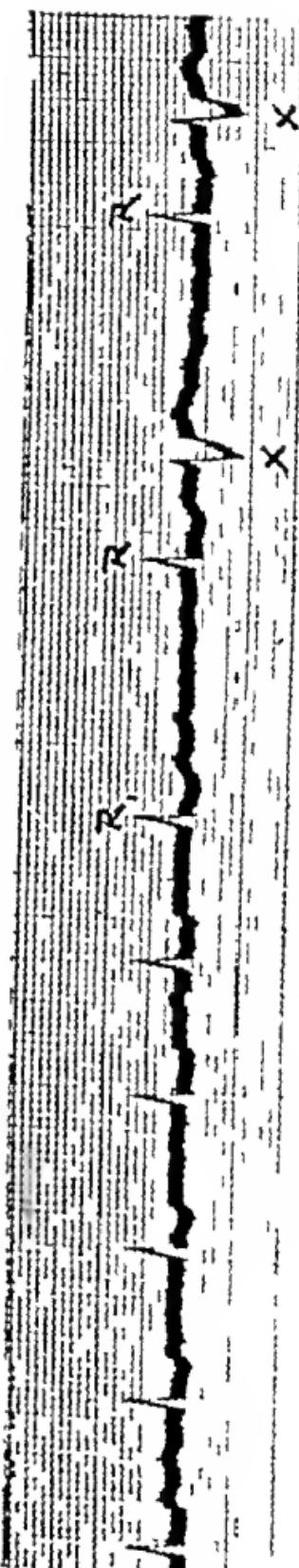


Fig. 205.—Auricular fibrillation showing transition to coupled rhythm.

of the heart muscle are modified by digitalis in the following particulars (1) the conduction is depressed, (2) the muscle fibers become more irritable, (3) the muscle tone is increased. With these facts in mind, let us examine the electrocardiogram (Fig 204) which shows the condition which you have found on examination this afternoon. First fix your attention on the waves designated by the letter R. These conform in contour to the waves of Fig 203, and are the normal ventricular contractions and occur rhythmically at a rate of 42 per minute. This means that digitalis has cut off all of the impulses coming from the auricles, and that the ventricles are contracting spontaneously and rhythmically quite independent of any auricular activity. In fact, we are dealing with a condition of complete heart block. The contractions of this series are strong and effective, and are those which produce the radial pulse waves which you counted at 42 per minute. Next look at the complexes designated by X. These are of ventricular contractions which have their origin not in the bundle of His, but in a point near the apex in the ventricular wall, hence these contractions are initiated in an abnormal point and sweep over the muscle-fibers by an abnormal path, recording themselves in the electrocardiogram in these bizarre complexes. These you recognize as ventricular extrasystoles. They are due to an abnormal irritability of the muscle caused by digitalis. One would expect that the ventricle contracting in such a manner would expel its contents less effectively than when the contraction proceeded in the orderly manner originally designed by nature, such is the case as you have already observed, for while you are able to count these contractions by auscultation of the heart, and thus estimated the heart rate at 84, not one of the extrasystoles could be detected in the radial artery, hence the rate there was found to be 42. As to the question of tone, we have no accurate method of measuring this fundamental property of heart muscle, but clinically it is quite evident that diastolic relaxation is much greater than that which furnishes an effective cardiac activity.

I want you to fix this "coupled rhythm" in your mind as an ~~important danger signal~~. It indicates that the patient is

getting too much digitalis. When this patient first came under observation fifteen months ago she received 45 minims of the tincture of digitalis a day and continued 30 minims a day for months with great benefit. Recently she has been taking only 15 minims a day, but even this was stopped at once when the "coupled rhythm" was discovered this morning. Figure 205 is an interesting record, as it was secured at the time of transition from her "good" rhythm to a period of "coupling." We can at once counteract part of the digitalis effect and probably abolish the coupling by the subcutaneous administration of atropin, $\frac{1}{8}$ grain, which will show its maximum effect within half an hour. This, however, will be at the expense of accelerating the heart to a rate of 100 or over. At times when a heart like this becomes oversusceptible to digitalis we can often improve matters by giving one of the bromids in combination with digitalis, and thus retain the slowing effect and at the same time do away with its irritant properties.

There is another condition which this patient presents and to which I wish to direct your attention, viz., her gastro-intestinal disturbances—the nausea, vomiting, and the formation of gas. This afternoon the symptoms were undoubtedly in part at least due to too much digitalis, but similar symptoms were present before she ever received digitalis. At those times these symptoms were due to the insufficient circulation and the consequent chronic passive congestion of her digestive apparatus. I have shown you her large pulsating liver and her large tender spleen, undoubtedly all the organs of her digestive tract are participating in this engorgement. In her earlier stay in the hospital these symptoms soon disappeared with the circulatory improvement under digitalis. The point I wish to make is that nausea and vomiting are not always due to the digitalis which is being administered, as many people, physicians included, seem to think, but if digitalis is persevered in it will in most instances correct the circulatory condition and thus cause the disappearance of the symptoms. The outlook for this young woman is not at all promising. We have been able to patch her up by regulating the heart activity on a number of occasions, ~~but~~ ^{but} ~~the~~ ^{the} time it

has become more difficult, and now digitalis, which she needs, fails us, since even very small doses produce toxic symptoms. It is quite possible that she may live for some months,¹ but I see no prospect of being able to improve her condition so that she will be able to leave her bed.

Recapitulation — 1. Rheumatic fever

2. Mitral stenosis and insufficiency

3. Auricular fibrillation, onset ten years after rheumatism

4. Followed by a marked degree of cardiac insufficiency

5. Gastro-intestinal disturbances due to chronic passive congestion.

6. Digitalis at first beneficial

7. Later causing complete heart-block and ventricular instability, as evidenced by the coupled rhythm

COMPARISONS OF THESE CASES

I have presented to you this afternoon two cases, both have mitral stenosis following acute infections, both have auricular fibrillation. One is doing well and the outlook for him is still favorable after eight years of continuous irregularity, the other is rapidly losing ground after only fifteen months of fibrillation. Why is the heart fairly efficient in the one case and totally inefficient in the other? I think we can find the answer in the quality of the ventricular muscle which these patients present. It is quite remarkable how extraordinarily well a patient may get along with fibrillating auricles provided the integrity of the ventricles is unimpaired. In the condition of fibrillation there is no co-ordinated contraction of the auricles. They act as reservoirs for the blood entering the heart much as a dilated vein might function, the only emptying force being the elasticity of their walls. If, as is usually possible, we can with digitalis effectively cut off the auricular haphazard impulses so that the ventricles are not too greatly disturbed these will go on doing their work quite efficiently. When, however, the ventricular myocardium is correspondingly damaged, the story is quite a

¹ This patient died in the hospital in March, 1919, two months after her presentation in the clinic.

different one. Compare the ventricular condition of our two cases. In the first there is very little evidence of ventricular damage, the muscle is not overirritable, blocking is not easily produced, there are no extrasystoles of ventricular origin, the muscle tone is good, and there is comparatively little dilatation. In the second case the muscle is so excitable that a complete block and multiple ventricular extrasystoles are produced by very small doses of digitalis, and loss of tone with great dilatation is an outstanding feature.

If you study your cases carefully you will find many minor features which will aid you in predicting what the future holds in store for these patients, but the most important questions will be, How much damage has been done to the *ventricles*? and What are their capabilities for performing their work?

CLINIC OF DR ALBERT R LAMB

PRESBYTERIAN HOSPITAL

NON-HEMOLYTIC STREPTOCOCCUS ENDOCARDITIS

Diagnosis Importance of Every Detail in Making a Correct Diagnosis. Course Management. Treatment. Subsequent Results Discussion of the Various Forms of Endocarditis

It is most important, gentlemen, that you should become familiar with the interesting group of cases of which today's patient is a striking representative. I emphasize this because I have found that each succeeding class is most hopelessly confused on this whole important subject, when its fundamentals are most simple. And today we shall endeavor to confine ourselves to fundamentals, leaving many of the finer points and disputed problems to the future. And this I take to be important because many of you are only too eager to discuss some still unsettled point in connection with a given disease before you have even the beginning of a clear-cut conception of the disease itself. Now, an enquiring mind, a doubting mind, an investigating mind is a priceless gift. If you have such a thing in your possession hold on to it and cultivate it. If you have not such a mind, try to develop one. The point I am making at the present time is that it is absolutely essential for each one of you to build first a foundation of complete understanding of the fundamentals for each disease or group of diseases. With such a foundation as a starting point you may then go ahead and enquire, doubt, read, investigate, and perhaps add something to existing knowledge. But, as I have said, at the present clinic we shall stick to foundations, and if we can build a proper one for this group of diseases the afternoon will not be wasted.

Case History—The patient, a young married woman of twenty-five, first presented herself for an opinion as to the advisability of having her tonsils removed on account of her previous attacks of rheumatism. One year before she had been feeling so exceptionally well and strong that she undertook a large number of courses in music. At the end of three months she felt weak and was obliged to drop some of her work, but did not stop entirely until another three months had passed, when, in addition to the weakness, she developed soreness in the small joints of her hands and in her finger-tips. One month later, or five months before admission, she had a severe attack of pain in the left upper quadrant of her abdomen. This persisted off and on for about a month, but has not troubled her since.

Four months ago she went to the country and felt better, but did not gain in strength as much as she had expected.

Three months ago she noticed, from time to time, a peculiar soreness in the tips of various fingers and toes. This soreness would last for a few days and then completely disappear. She says that it felt exactly as if she had gotten a thorn in her finger. During this time she noticed that her color was not so good, although she had always been pale.

For the past two months she thinks that she has had fever at times, but no temperatures have been taken. There has been a gradual loss of weight and for several weeks real night-sweats. From time to time she has had transient joint pains and more or less palpitation, with no other signs of cardiac decompensation.

Her family history is unimportant. Her habits are good. She lives in New York in the winter studying music and spends the summers on a farm. Has always been very careful about eating, drinking, exercise, and sleep. She has been married five years, but has had no children on account of her heart.

She has had measles, whooping-cough, and chicken-pox, but never scarlet fever or diphtheria. Has never been subject to sore throat, but has always had more or less nasal catarrh, with blood frequently present in the secretions. Tonsils were partially removed at eight.

At ten years of age she had her appendix removed. Shortly

after this she was kicked in the forehead and trampled on by a horse, resulting in a dislocated shoulder, "water on the knee," and dislocated fingers. Had acute otitis media with paracentesis of the drum at nineteen.

For the past seven years she has had more or less indigestion and gas associated with constipation.

At the age of twelve the patient had her first attack of rheumatic fever. She had pain in all of her joints and muscles and was confined to bed. The pain disappeared promptly, but the fever continued for two or three months. Her doctor told the family at this time that her heart was affected. The following year she had a mild attack of rheumatic fever lasting for three weeks. Seven years ago there was a third severe attack lasting for three months. Since then there has been no recurrence.

Such, gentlemen, was the story when this young woman presented herself to see if it were advisable to have her tonsils removed. She had been told by a good laryngologist that her tonsils were diseased and that they should come out if her general condition did not contraindicate it. Now I trust that it is entirely superfluous for me to utter a warning against anyone of you giving advice on such a serious question without the most careful consideration of all the facts in the case. In the first place, we must estimate the degree of cardiac damage and reach a decision as to whether the heart is well compensated and able to stand the operation. In the second place, we are face to face with the problem of why this patient has been failing in health for the past nine months with certain symptoms which we shall take up presently. Is this due to chronic infection from the tonsils or is it something else? In other words, we must not jump to the point of giving advice until we have a very clear-cut idea of the diagnosis and the patient's condition. There are no short-cuts in medicine. If you learn nothing else today, please carry away a fixed determination to go into each case completely and carefully, letting no fact escape you.

Therefore in this case we shall allow the question of tonsillectomy to wait upon the diagnosis and prognosis, and we shall find that it then answers itself.

Now, we have this patient's history up to the time of her admission to the hospital one week ago, and it has always seemed well to me to pause in any case after a history has been obtained and consider for a time what the possibilities are. For we are all too prone to take a history and make a physical examination in a routine way, and then find to our surprise that the diagnosis is still in the dark. The history suggests or should suggest certain possibilities, and by keeping these in mind during our physical examination we are much more apt to observe things which we might otherwise overlook. I know of nothing more humiliating than to have a really skilled observer go over a case and point out things which we have failed to see. You have all doubtless experienced this. But, fortunately, clever observation is largely a question of training and many things are seen because they are looked for. Now, without going into any lengthy discussion, let us see what the possibilities are in this case, then let us see how the physical examination alters these possibilities or adds new ones, and we shall find ourselves in a position to attempt a proper correlation of the known facts. Will anyone suggest a possible explanation of the patient's symptoms?

STUDENT Pulmonary tuberculosis

DR LAMB Yes, this must be kept in mind. The patient has been gradually failing in weight and strength, she has had definite night-sweats, and thinks she has had fever at times. I have asked her about cough, but she denies having had one. Many of these cases do have some cough, probably from slight cardiac decompensation, so that you may easily understand the frequency with which this disease is mistaken for pulmonary tuberculosis. If there be a pulmonary embolus with consequent hemoptysis, the mistake is even easier. What other diagnosis would you suggest?

STUDENT Articular rheumatism

DR LAMB Gentlemen, I wish that today, once and for all, you would give up the term "articular rheumatism." The proper term is "rheumatic fever." And it is much more important here than in most diseases which have been incorrectly named, for it serves to focus the attention constantly upon the joints, and

makes one forget that rheumatic fever is just as much a disease of the endocardium, myocardium, and pericardium as it is of the articulations, and that there may be associated with it tonsillitis, cutaneous lesions, chorea, pleurisy, and muscular involvement. Now, just what do you mean by saying that this may be a case of rheumatic fever? Certainly, it is not the usual picture of that disease. The patient who has had three definite attacks of rheumatic fever will tell you that she is not suffering from a similar disease at the present time.

STUDENT I meant rheumatic endocarditis.

DR. LAMB Now we are becoming definite. And let us always strive to be definite in medicine where we can, for, goodness knows, there is enough that is indefinite. Don't speak loosely of a case as a "myocardial affair," or as "some renal condition." Say definitely what you mean in terms of the pathologic picture. This is good training and will keep you from making many ridiculous mistakes. Rheumatic endocarditis is a most important consideration in this case. We know that she has had rheumatic fever in the past with cardiac damage, and much of her story can be explained by assuming that she has been suffering from recurring endocardial infection with whatever it is that causes rheumatic fever. We shall go into it more at length presently. Are there any other suggestions?

STUDENT Chronic sepsis from some focal infection.

DR. LAMB Such a condition must certainly be considered, and in going over the case we must investigate all possible sites for such infection most carefully. And, in particular, we must remember that she once had ear trouble, that there has been a chronic nasal catarrh, and that she comes to us on account of her tonsils. Most of the cases of rheumatic fever have tonsillitis or sore throat, and as this usually precedes the attack it is not surprising that most medical men consider the tonsils as the main portal of entry for the virus of rheumatic fever. While most suggestive, please bear in mind that this is not yet proved. Nor do we know whether with chronic tonsillitis there is a chronic rheumatic fever. We do know, however, that one may have an arthritis from such a source caused by streptococci.

which we believe to be a thing distinct from rheumatic fever and which should be called by its proper name, "streptococcus arthritus" We also know that in some cases of true rheumatic fever, streptococci may be isolated from the blood and affected joints There are those, as you know, who are convinced that in these organisms we have the definite cause of rheumatic fever We do not consider the evidence as convincing and prefer to look at these streptococci, occurring in only a small number of the rheumatic cases, as secondary invaders Our viewpoint is, that, on the one hand, we have our cases of true rheumatic fever, and, on the other, our cases of true streptococcus arthritis, with many borderline cases whose position it is as yet impossible to establish There is another important disease which no one has mentioned

STUDENT Malignant endocarditis

DR LAMB Just what do you mean by that?

STUDENT Streptococcus viridans (infection of the heart valves)

DR LAMB Here again we are using terms incorrectly, and as a considerable part of the difficulty in this whole subject is one of terms, I must keep insisting upon your using the correct ones Almost any infection of the heart valves with bacteria may be a malignant endocarditis Infection with the Streptococcus viridans, so called, is only one variety, but a most important one And in this case it must be considered most carefully

Now we have seen what some of the possibilities are in this case Perhaps, in addition, we should consider some grave form of anemia or a simple, mildly incompetent heart, but neither one of these diagnoses explains the possible fever, night-sweats, pain in the left upper quadrant, transient joint pains, and sore fingers and toes One other thing I consider of importance in any case, and that is, to remember that it may, after all, be one of unusual character, in other words, we may be dealing with a disease which we do not suspect By remembering this in every case we are always on the lookout for something which does not fit into our explanation of the case and which may serve as a clue in directing us along the right path

It has always seemed to me best to approach any case in the

above manner. Now this does not mean that we necessarily sit down after taking the history and go into all these details. They suggest themselves as we go along, provided that we are trained in the right way of thinking, and by the time we approach the physical examination we have, or should have, in our mind an orderly idea of the possibilities of the case. But you will need really to sit down and think over each case in this way for some time to come in order to train yourselves properly. And I believe that if you do this you will be well repaid.

And so as we finish this patient's history the case appeals to us somewhat as follows. There were in the past several attacks of rheumatic fever which caused cardiac damage. Now there is nothing to prevent a person with a damaged heart from having some entirely independent disease. But such a history is, nevertheless, important, and we shall do well never to neglect it. It is the background of the case which we must keep in mind as we go along. With such a background, this patient has been gradually failing in health for the past nine months. The symptoms of weakness, loss of weight, probable fever, night-sweats, and pallor are what we might expect from some low grade infection. This infection might be in the nature of one of the things which we have briefly considered. But the pain in the left upper quadrant of the abdomen and the sore fingers and toes are things of the greatest importance, and should go a long way toward making a correct diagnosis if we know how to interpret them properly. It is, therefore, time to turn to an examination of the patient to see what confirmatory or contradictory evidence this examination may throw upon the case.

Physical Examination—I think that you will at once be struck by the fact that this patient looks sick. Her eyes have the peculiar heavy appearance of one who is not well and there is an unmistakable waxy pallor. She is not dull or apathetic. On the contrary, there is a very noticeable alertness amounting almost to apprehension, such as one sees in certain types of disease. She is fairly well nourished, and the loss of weight which she has mentioned is certainly not very striking. There is no dyspnea or cyanosis. The nose, ears, sinuses, antra, and teeth show no

evidence of any foci of infection. This has been checked up by good men, and we have their assurance that these common sites for focal infection can be eliminated in this case. There are no rashes, jaundice, or eruption of any kind. The pupils are regular in outline, equal, and react promptly to light and accommodation. The tongue is moist and clean. Her tonsils are really quite small, but are adherent and somewhat cryptic, but it is not possible to express anything from these crypts. The superficial lymph-glands are not enlarged. Her thyroid is just barely visible and palpable. As you know, this is not at all uncommon in young women. There are no signs to indicate hyperthyroidism. As we have considered pulmonary tuberculosis as one of the possibilities, a very careful examination of the lungs is indicated. This has been made by several examiners and all report the lungs perfectly clear throughout. The cardiac apex is in the fifth space, 8.5 cm to the left, and the left limit of cardiac dulness is 10 cm to the left in the same space. No enlargement is made out to the right. The first sound at the apex is very loud, sharp, and valvular in quality. It is followed immediately by a loud, high-pitched, blowing systolic murmur heard best just within the apex, but very audible over the whole precordium. The second sound is sharply accentuated. At the base the first sound in the aortic area is partly obscured by a blowing murmur, followed by a soft second sound. The action is perfectly regular.

The abdomen shows nothing abnormal except a firm smooth spleen which descends fully 3 cm below the costal margin on inspiration. The patient says that it was in this region that she had the pain previously described as in the left upper quadrant.

There is no edema and all of her reflexes are quite normal. The joints all appear normal at the present time. As you will notice, the tip of the right ring-finger is quite tender. It is purplish blue in color and is quite cold to the touch. Her temperature is 102.8° F., pulse 100, and respirations 24. Blood-pressure, 112/68. As the patient can give us no more information at this time we will excuse her.

We are now in a position to consider all the facts in the case available up to the present time. No matter what your

method of procedure has been up to this point, it is now up to you, before proceeding further, to argue out your tentative diagnosis or diagnoses. This should always be done in every case at the completion of your first examination. The headings "Conclusions" should be just as intrinsic a part of your case records as should the "History" and "Physical Examination." Under this heading you should state your impressions of the case with a logical analysis leading to your diagnosis. In addition, you should state your proposed line of procedure to clinch the diagnosis, your idea of the prognosis, and the line of treatment to be instituted. Where you find it impossible to decide between two or more diagnoses, I cannot too strongly recommend that you place figures of percentage of probability after each diagnosis. It is, indeed, most instructive and often most startling to look over these preliminary conclusions after a case is fully developed. The facts are there as you saw them and there is no possible way of crawling out of them. I know of nothing that is of greater assistance in developing your medical ability and in training your mind in the habit of clear and logical thinking.

In this connection let me urge each one of you to keep a card labelled "Mistakes" in your file. I trust that you will all have enough such cases to keep you duly humble, but not humiliated. Of one thing I am sure If you will follow the advice just given you will cut down the number of your mistakes most appreciably.

those demanding the largest number of additional laboratory examinations. Of course, in a hospital one may have all manner of laboratory examinations carried out. But that is no reason for doing so. The laboratories are overworked, and we should all aid in making them the more effective by cutting down all unnecessary work. In private practice most people cannot afford innumerable laboratory examinations. But to my mind, even more important than either one of these considerations is the very pernicious resultant deterioration in our powers of observation and logical medical reasoning from relying on the laboratory to make our diagnoses for us. Let us ask for only such laboratory work as is distinctly indicated, let us ask for it in its order of relative importance, let us be chagrined rather than pleased if the laboratory turns up some unexpected diagnosis, and let us not cover up our inability to arrive at a diagnosis by asking for some piece of laboratory work as if it were the fundamental point in the case. Naturally, I do not here refer to the very obscure cases where sooner or later we have to call into play every resource of the hospital, nor to the special cases which are being worked up from the standpoint of scientific research. In general work the laboratory should be used to establish or refute the conclusions arrived at clinically, and should not be used as a substitute for a little gray matter and analysis on our part.

But let us return to our immediate task. Let us see if the examination has, in the first place, helped to rule out any of the possibilities which we considered. It is best to approach the problem from this standpoint, for it narrows it and allows us to concentrate upon a few things. Unless some new fact develops as we go on with the case, it seems to me that we are fairly safe in ruling out pulmonary tuberculosis. The lungs are quite clear and there are several facts in the case which are not explained by it.

In the same way we may be permitted to side track the diagnosis of chronic sepsis from focal infection, for we have been able to discover no such focus, and this inability has been confirmed by x-rays which I have not previously mentioned. Now, of course, it is not positively ruled out, and we may have to keep it

in the back of our minds until we see if the case cannot be completely cleared up on some other grounds

We now know that this patient has fever. It is, therefore, almost certain that we have to do with something more than a mildly incompetent heart. The blood count is really part of any physical examination. In this case it was as follows: R. B. C., 4,200,000, Hg., 70 per cent., W. B. C., 11,200, polymorphonuclears, 79 per cent., lymphocytes, 21 per cent. We have, therefore, no grave form of anemia to deal with. It is simply a moderate secondary anemia, which, however, is belied by the patient's appearance, and a slight leukocytosis, all of which would fit in with our idea of a low grade infection. I would remind you, however, that occasionally there is real difficulty in the differential diagnosis between this disease and pernicious anemia. While not of importance in the differential diagnosis in this particular case, you must remember that the gradual onset with fever and a palpable spleen quite commonly attracts the diagnosis of typhoid fever. In fact, these cases are most often mistaken for either typhoid or tuberculosis.

Thus our problem seems to have narrowed down to the constantly recurring one of heart, rheumatism, and bacterial infection of the heart valves, always remembering and being on the lookout for something quite unexpected. If this problem is clear in your minds you form a great exception to all recent classes.

The first fundamental point is the distinction between endocarditis and chronic cardiac valvular disease. The former is an active process which is going on in the endocardium, usually of the heart valves. Now it may be going on acutely or subacutely or chronically. The point is that it is going on. The latter is an end result, a mechanical defect. There is no active process going on. Originally, this mechanical defect may have been caused by rheumatic fever, syphilis, or possibly some bacterial infection from which recovery took place. At the time it was taking place we had a rheumatic or syphilitic or bacterial endocarditis. When these infections ceased we passed to the mechanical results of these previously active infections and had a chronic cardiac valvular disease. When known it is well to indicate the

original cause of this. Thus we can speak of a chronic cardiac valvular disease of rheumatic origin or of bacterial origin. Atherosclerosis, congenital anomalies, or trauma may also be the cause of mechanical defects. In our case of today we know that she had a rheumatic endocarditis at the age of twelve with probable recurrences at thirteen and eighteen. If we had seen her a year ago when she was well and strong we would have had little hesitation in saying that she had a chronic cardiac valvular disease. There was then no question of an endocarditis. Today she still has her chronic cardiac valvular disease. She will always have it. The question is whether, in addition, she now has an active process going on in the heart valves, an endocarditis, and if so, what its nature is. Now, it is not at all easy to decide in a given case whether we are dealing with the one condition or the other. But stating a problem clearly goes part way toward solving it, and if you begin to approach your cases in this manner you will gradually develop your criteria of distinction. It is, of course, quite impossible to state at what exact moment an endocarditis ceases to be one and becomes a simple mechanical defect. We all know how the mitral valve, affected by rheumatism, becomes gradually more and more stenotic, and it is an open question whether this is all simply scarring or as to whether there is not a constant slight rheumatic infection going on all the time. We cannot distinguish between the two conditions as finely as this. Where we have a valve defect without symptoms which we can recognize we are safe in calling the condition a simple chronic cardiac valvular disease. It is quite similar to the problem of deciding when an active tuberculous focus in the lung becomes a healed lesion.

The second point of great importance is for you to divest yourselves of the various terms which you do not understand and which are responsible for much of your confusion. It is just as essential that you do this here as in the case of nephritis. There is this difference, however. In the latter case there is no etiologic classification yet possible. With our present problem there is, and this is the ideal method of nomenclature. You have all heard these various terms. They include malignant

endocarditis, infectious endocarditis, ulcerative endocarditis, septic endocarditis, endocarditis lenta, subacute bacterial endocarditis, etc. As you will notice, some of these terms try to picture the pathologic picture, others attempt to indicate the prognosis, while still others approach an etiologic basis. Let us sweep them all overboard for the present and see what the facts really are. The valves of any heart may become the site of infection by practically any of the micro-organisms. In almost all instances such an infection is engrafted upon a previously damaged valve, that is, upon a chronic cardiac valvular disease. And we thus have an endocarditis. It may be acute, lasting only for days, or subacute, lasting for weeks, or chronic, in the sense of lasting for months. These terms indicating the course are really not essential, but it is essential to introduce the name of the infecting organism. Thus we may have a pneumococcus endocarditis, a *Staphylococcus aureus* endocarditis, a streptococcus endocarditis, in fact, any kind of endocarditis. And so we have the ideal etiologic terminology. Any one of these is malignant, infectious, and septic in the sense in which these terms are used. Some may be ulcerative, but this is a pathologic picture. Most are not. The terms "endocarditis lenta" and "subacute bacterial endocarditis" have been reserved for the cases due to the so-called *Streptococcus viridans*. But there is no more reason for continuing these terms than there would be in inventing new descriptive ones for some of the other bacterial endocarditides. The point is that we have very definite names according to the micro-organisms involved, and that keeps the whole thing straight. It is only necessary to remember and to be able to place these various other names in order that we may understand what some people are talking about when they use them.

Now, naturally, the various endocarditides vary more or less according to the causative organism. Some of the forms are well understood, others much less so. Today it is not our purpose to go into these differential features, interesting though they be. If you can only get a proper framework for the whole subject now it will be comparatively simple for you to add to the structure as you go along. As you meet each new type, you can

study it, read about it, and perhaps do some real investigation upon it. It will all fit into your general scheme in an orderly manner.

About one type you know or should know a great deal, namely, syphilitic endocarditis. According to our scheme this is a Spirochæta pallida endocarditis, but there is absolutely no harm in using the other term, as it means the same thing. This form, more particularly the aortitis part of it, is so well known now that it is generally considered apart from the bacterial endocarditides, although it belongs there just as truly as do any of the others.

Another well-recognized form and one which directly concerns us today is the rheumatic endocarditis. It, too, is almost always separately considered, although, without doubt, it belongs in this general group in spite of the fact that the causative agent is probably still unknown. The term "rheumatic" is used in place of the micro-organism just as the term "syphilitic" is used in place of Spirochæta pallida. It has a well-recognized pathology, and as it is, by all odds, the most common cause of endocarditis it is of the greatest importance. We could easily spend several afternoons on rheumatic fever and its various manifestations, but for the present we must content ourselves with a consideration of some of the important points concerning rheumatic endocarditis. Clinically, there is no way of deciding definitely whether a case is one of rheumatic endocarditis or not. That is, there is no decisive test, such as a complement-fixation reaction or the isolation of the causative agent by blood-culture. But in most cases this form of endocarditis is associated with a definite attack of rheumatic fever with all of its characteristic features. None of us should fail to recognize such a case. In the second place, we frequently see cases where the joint symptoms rapidly clear up, but where the endocarditis progresses. These cases are not always recognized. You will do well to think a bit about them. If a patient has recovered from the acute symptoms of rheumatic fever, but still runs a slight temperature, has a pulse elevated out of proportion to this temperature, and develops a heart murmur, changes the quality of an

existing murmur or develops an additional one, I think that you are justified in considering that that patient has rheumatic endocarditis

Obviously, our patient does not fit into either of the above groups. Our troubles begin when we reach the third and most tantalizing group, that in which there is a true rheumatic endocarditis with only the most transient or fleeting joint symptoms or with none at all. No one knows how large this group is. We do know, however, that it exists, and I suspect that it is much more important than most of us realize. You have all, I am sure, been impressed by the fact that the cardiac damage does not run in proportion to the severity of the attack of rheumatic fever. You have seen most extensive damage from the mildest cases, and the reverse. Some of you have seen a case recently in the wards. This boy entered the hospital with a considerable fever and high pulse. It was quite unexplained until the second day, when he developed a transient pericarditis. This disappeared the same day, but his temperature did not return to normal, and in the course of a short time he developed heart murmurs and was ill for some time. He had never had rheumatism and at no time during his course did he have any joint symptoms. Repeated blood-cultures were negative. Now, as I have said, we have no means at our disposal to settle this question, but in the absence of any positive evidence pointing to any other type of infection I feel that we are justified in considering this a case of rheumatic endocarditis and pericarditis. And please remember in this connection that although we are emphasizing the endocarditis, in practically every case of rheumatic infection all parts of the heart are affected. Sufficient importance has not been attached to these cases. Otherwise we would not hear so much about cases of idiopathic mitral stenosis. A very large number of such cases must be examples of rheumatic endocarditis which went unrecognized because of the absence of joint symptoms. This is a fruitful field for investigation, but the question will probably not be settled until the cause of rheumatism is surely discovered.

The only other group of endocarditis which interests us today

is probably the best known of them all, and yet it was only a few years ago that many consultants made or enhanced their reputations by its diagnosis. I believe that each and every one of you should be able to recognize it by the time you leave school. As a matter of fact, I fear that there is more danger of your making the diagnosis when the disease does not exist than there is in your missing it when it does. It is by no means rare, and you will have the opportunity of seeing several cases before you graduate. I refer to the so-called *Streptococcus viridans* endocarditis. This is not the right name for these streptococci, for they do not all produce green on blood-agar-plates, as the name *viridans* would indicate. Also, you should not use the term "*Streptococcus mitior*," introduced by Schottmüller as descriptive of the organism's low virulence, as other streptococci may be of low virulence. They are, however, all non-hemolytic, and this term is, therefore, the correct one. The disease has very definite characteristics which distinguish it from the various other varieties of bacterial endocarditis, except that due to the influenza bacillus. These two forms are indistinguishable clinically. Influenza endocarditis is quite rare. Nephritis is supposed to be a frequent complication. But the distinction is made between the two only by blood-culture. They differ from all the other forms in being strikingly subacute or chronic. One should have no more difficulty in diagnosing non-hemolytic *streptococcus* endocarditis than one should have in recognizing a well-marked case of typhoid fever. We shall take up its characteristics as we proceed with the analysis of our case.

The way is now clear for us to round up our case. The first thing necessary in arriving at any diagnosis is correct observation. That is, if we have left out important points in the history or examination the whole thing is as hopeless as a picture puzzle from which many pieces are missing. So to start with, let us assume that we have skipped nothing of real importance. In the second place, the symptoms and signs point more or less clearly to one or more conditions. And in this regard we have narrowed our problem down to a differential diagnosis between rheumatic and some other form of bacterial endocarditis, probably non-

hemolytic streptococcus. And this is a constantly recurring problem, and it is here that you will make your mistakes in diagnosing non hemolytic endocarditis when it does not exist. Your problem in these cases is this. You have a case of chronic cardiac valvular disease who is well and running a temperature. The first thing to do is to be sure that this patient has not some disease entirely unassociated with the heart valves. As I have previously said, there is nothing to prevent such a person having typhoid fever or malaria or anything else. With such possibilities out of the way the second thing to do is to consider whether you are dealing with the fever so often seen with a badly compensated heart. If there is no decompensation this possibility automatically disappears. If there is decompensation we know that it is possible for the patient to run a temperature until compensation is re-established. I do not think we know what this fever is due to. In some cases it is from small patches of bronchopneumonia, or infarcts, or infections which are analogous to terminal infections. The decision here between fever with decompensation and some form of bacterial endocarditis is not easy. But observation and blood-cultures usually suffice to settle the question. At any rate, it does not concern us with our present patient. If we can thus rule out the above conditions, we come to rheumatic versus some form of bacterial endocarditis, and the former can be decided upon only by elimination and by the things in the history and examination which do not fit.

And these things which do not fit form the third important step in diagnosis. In any case a certain number of the symptoms and signs are completely explained by our tentative diagnosis. There are usually, however, one or more things which are not explained. They do not fit into the picture. And I am in the habit of asking you to ring these misfits and to consider them most carefully, for in a great many cases it is the proper appraisal of these facts which do not fit that spells success or failure in solving the problem. We always like to explain all the facts upon the basis of one disease if possible. Sometimes it is undoubtedly wise not to do so, but I am positive of one thing, and that is that you will make a serious mistake if you carelessly

introduce some new disease or complication to fit each unexplained fact

Now with our case, most of the facts can be explained by rheumatic endocarditis, but there are some which cannot. The pain in the left upper quadrant is one misfit. You might explain it as a pleurisy, which is not infrequent in rheumatism, but we have investigated it more closely and find that it was too low for a pleural pain. As a matter of fact, it was over the spleen, which we find very distinctly enlarged. There is nothing in her past history to account for an enlarged spleen, and it is certainly not the spleen of chronic passive congestion, for she has not this condition. We shall not go far wrong in interpreting this symptom and sign as due to an infarct. Now an infarct from rheumatic endocarditis practically does not occur. If the heart is decom pensated there may be a thrombus in one of the auricles or even in one of the ventricles with a resultant embolus, but this is a result of the decompensation and not of the rheumatic endocarditis. Embolism is no part of rheumatic endocarditis. On the other hand, it is exceedingly common in non-hemolytic streptococcus endocarditis. As our patient is not decompensated we have gone a long way toward ruling out rheumatic endocarditis by this one misfit.

The tender toes and fingers, with the appearance which you have seen in one of them, are no part of rheumatic endocarditis and are classical for the other conditions, so that I feel that in conjunction with the above we can dismiss rheumatic endocarditis.

Therefore, by the process of exclusion we have arrived at the diagnosis of non-hemolytic streptococcus endocarditis, or of some condition of which we have not even thought. Now let us see if the facts all fit the former, or if we have some things still unexplained.

Practically every case of this disease is engrafted upon a pre-existing chronic cardiac valvular disease, usually of rheumatic origin. This is an essential background for the diagnosis. And so we start right, for our patient has had such a condition for a number of years. And here I must interject a warning. Always

remember that a patient with a valvular defect is a potential candidate for a non hemolytic streptococcus endocarditis. It must always enter into your prognosis of any case of chronic cardiac valvular disease, though you do not need to terrify the patient with your idea, and you must always remember its possibility when such a patient is failing in health. If you will always bear this in mind you will not be apt to miss any case, for, as the late Dr E G Janeway was accustomed to say, whenever a rare disease turned up unexpectedly, "I believe we could make that diagnosis if we could only remember at the time we saw it that such a disease existed."

The onset of the disease is as insidious as the disease itself. The patient has simply not been feeling as well as usual since "before Christmas" or since "last fall." Probably most of these patients have been suffering from the disease for weeks before they even realize that they are not well. Our patient had been failing since some time about nine months before. She had tender fingers six months ago and her splenic infarct five months ago. As both of these symptoms are characteristic of the disease, we can feel almost certain that she was well in its clutches some six months before she felt it necessary to consult a physician, and then not because she considered herself seriously ill, but simply to find out if she would not be better off without her tonsils.

From the time these cases are first seen they have fever. It is very slight at first, often not more than half a degree or a degree in the afternoon. Some afternoons it may not be elevated at all. Such a low-grade fever may go on for several weeks before it begins rather wider swings. As the disease progresses there may be four or five degrees between the high and the low. We have no temperature records for our patient, but she has volunteered the information that she thinks she had fever at times, and for the past week it has run between 99° and 101° F.

Loss of weight and strength are constantly present and progressive, as in our case. They do not lose rapidly, but they do it persistently.

There is a slowly developing anemia. Like the rest of the

disease, it is insidiously progressive. In the terminal stages it is usually most extreme. It is practically uninfluenced by iron and arsenic, but for a time is remarkably benefited by transfusions. Our case is not so anemic according to her blood count as we have a right to expect. As a matter of fact, I think that you will agree with me that her appearance belies her count, and that we may, therefore, doubt its accuracy. We shall have it repeated.

None of these patients shows a normal color. Many have a very characteristic *café-au-lait* color, which is most suggestive. But this is not essential. The others have an appearance much like that you have just seen. It is a waxy, almost transparent pallor, either quite white or with a dirty tinge bordering on the *café-au-lait*.

Night-sweats are quite common at almost any time in the course, as are chilly sensations. Real chills are usually seen only in the later stages.

It is not at all unusual to have mild, fleeting joint pains, as in this case, and also pain, soreness, and stiffness in various muscles, but the joint pains do not approach in severity those of rheumatic fever.

Thus far the symptoms are not characteristic. They are simply what one would expect with many a low-grade infection, and from them one cannot make a diagnosis. The important point is that they occur upon the background of a chronic cardiac valvular disease. This should make us extremely suspicious, and, being suspicious, we can look more closely for symptoms and signs which are bordering upon the pathognomonic. It is often a fact that the symptoms which are of the highest significance are not volunteered by the patient. They may not have attracted attention because of their trivial nature or they may have been forgotten. Thus in our case the sore fingers and toes were not mentioned until the question was definitely asked, as the patient had interpreted them as part of what she considered rheumatic manifestations.

And yet these sore fingers and toes are among the highly characteristic symptoms of the disease. They occur at some

time in the course of most of the cases. They come on suddenly, are quite painful, feel numb or prickly and cold, have the purplish-blue color with whiteness of the rest of the tip, as you have seen, and clear up in the course of a few days. It is not usual to find them in any other condition, so that if they are definitely present in conjunction with such other symptoms, as we have found in this case, they should go a long way toward determining the diagnosis.

The pain in the splenic region with the presence of an easily palpable, hard spleen is another thing of great significance. Like the sore fingers, this comes on quite suddenly, but it is apt to last for a considerable length of time. The pain is sharp and stabbing in character and is generally made worse by inspiration. As you know, this is due to an embolus, as are the sore fingers and toes. One frequently has to ask for this symptom, as the patient may well skip it or simply remark that she had some pain in her stomach. So you see that it is absolutely necessary for you to know that these facts are among the fundamentals in the diagnosis of this group of diseases. If the patient has had pain in "the stomach" it is up to you to locate it exactly, and if it be in the splenic region and be corroborated by a palpable spleen you have facts which are essential in the diagnosis.

Now a splenic infarct does not necessarily mean non hemolytic streptococcus endocarditis or any other form of endocarditis. They are fairly common, as you will note if you watch for them. They mean that somewhere in the body there is a thrombus located so that it is possible for an embolus to reach the spleen. This source is usually the left side of the heart. It may be from vegetations on the valves or from a thrombus in one of the chambers. Now, as you are aware, it is most extremely unusual to have a thrombus in either the auricle or ventricle, except where there is a considerable degree of decompensation or in the rare cases of coronary thrombosis. If there is real decompensation and an infarct occurs one should hesitate a long time before ascribing it to the form of endocarditis which we are considering. It would help not at all in the diagnosis, which would have to be made by other means. We need scarcely go

into the cases of coronary thrombosis. If such decompensation be not present and we have an infarct we may well think seriously of an endocarditis, of course, remembering other possible sources for thrombi. One thing we know is that rheumatic endocarditis *per se* does not cause embolic phenomena, or at best such an occurrence must be so rare as to justify us in practically precluding it. Most of the other forms of bacterial endocarditis do cause emboli, and especially the one which we are discussing. So that the occurrence of emboli is an important differential point in the diagnosis between rheumatic and non-hemolytic streptococcus endocarditis.

And what has been said about splenic infarcts and the sore fingers and toes applies equally to other embolic phenomena, such as infarction of the kidney, cerebral embolus, and embolic aneurysms. In a suspected case of bacterial endocarditis one should keep a sharp watch for emboli of any organ. The point is that those of the fingers and toes and spleen are the most common and most readily recognized. A cerebral embolus speaks for itself. Renal infarcts are quite common. In addition to the pain in the costovertebral angle which is quite commonly absent, the striking thing is the presence of red blood-cells in the urine.

We have thus far considered all the symptoms and signs which this case presents. We find that there are no misfits. That is, everything can be explained by the diagnosis which we have arrived at, and by it better than by any other. There is not a single clue pointing in any other direction, so I feel that we are safe in discarding the possibility of some quite unthought of condition.

As you will remember, further back we discussed some of the steps necessary in arriving at any diagnosis. At this point it is well to point out a fourth necessity. And that is to see if there are missing signs and symptoms which are important in the diagnosis, and if there are such things absent, are they important enough to make our conclusions impossible. Of course, none of you expects every symptom and sign of a given disease to be present in every case of that disease. But it might well happen and frequently does that enough important ones are absent to

make the diagnosis unjustifiable. As a matter of fact, some of the men on the staff who had the opportunity of observing this case would not commit themselves on account of the absence of at least one such point. Can any one suggest essential missing data?

STUDENT Petechiae

DR. LAMB That is the point. Petechiae are most important in this disease. I doubt if there are any cases in which they do not appear at some time in the course. Their continued absence should make one very suspicious. But they must be looked for, not casually, but with the most painstaking care. Every part of the body must be examined every day, and you must remember to look in the mouth and in every part of the conjunctiva and also at the eye-grounds. Up to the present time, in spite of the most careful search, we have failed to find a single petechia. And yet, gentlemen, I feel that we have enough positive evidence to justify our diagnosis clinically, and I feel certain that petechiae will be found. And I want to urge you to have the courage of your diagnostic convictions. If you have argued a case out carefully and arrived at a conclusion that seems justified, do not be afraid to say that that is what you think the case is. Also I want to urge you to take every opportunity to become familiar with the appearance of petechiae. I am constantly surprised at the number of men who do not know them when they see them. I see no use in going into any description of these spots. The only way to become familiar with them is to see and study them. Most of you confuse them with telangiectases. Study these, too, for you must know the difference. Petechiae are not limited to the form of bacterial endocarditis which we are considering. They may be present in many conditions, and it is well to remember that they may occur in the late stages of chronic cardiac valvular disease. It is not unusual to see men fooled into a diagnosis of bacterial endocarditis by their presence in such a condition, and I think that you will make more mistakes in diagnosing non hemolytic streptococcus endocarditis simply because petechiae are present than you will in failing to diagnose it because they are absent. If the case shapes up right

otherwise, however, they are a most important determining factor. Are there any other important points to consider?

STUDENT How about tenderness of the sternum?

DR. LAMB We do not find this sign in many of the cases, and it is possible to elicit it in cases other than those of this disease. Therefore, we have never rated it as an important sign. It is interesting and worth looking for, but I do not believe that it should influence us much in our decision. It has not been present in this case.

From a clinical standpoint I feel that we have now covered the essential points of the subject. And we come to the determining point in diagnosis, namely, clinching it by means of the laboratory. If you could have only one piece of laboratory work carried out, what would you request?

STUDENT A blood-culture

DR. LAMB Yes, that is absolutely essential. And the second thing you would request would be another blood-culture to check the first, if it has been positive, or to endeavor to get a positive one if it has been negative. And if negative and your analysis of the case has led you to the decision which we have here reached, you should keep on taking blood-cultures until the case is settled in your mind. For as you know, one may obtain several negative cultures before having a positive one. Also remember that where you have several negative cultures in a case that still seems to be one of non-hemolytic streptococcus endocarditis you may be dealing with a case of infection by the influenza bacillus. Remembering this, you can take appropriate measures to grow this organism. Your blood-culture should be plated, as it is a more certain means of getting positive results where there are but few organisms, and it also gives one, for comparative purposes, a means of estimating the degree of bacteriemia through the count of the colonies.

A blood-culture was taken on our patient on admission, and the plates showed an average of 41 colonies per cubic centimeter of a non-hemolytic streptococcus, which in this case was a viridans. This was confirmed. Thus it seems to me that we have tracked down this case to an unescapable conclusion. We have

established the diagnosis, there is no present reason to believe that there is any complication, and I think you will agree with me that the question of tonsillectomy has been automatically settled.

Now the patient and family are not especially interested in all of our technical discussions. They want to know in simple language what the trouble is, but what they are mainly concerned about are the chances for recovery and the length of time the illness will last. This brings us to the much neglected field of prognosis. We have not the time to go into the general principles of this question. A whole afternoon could be thus spent most profitably. In this disease you must know, in the first place, that it lasts for a long time, from many months up to two years. Therefore, you have to prepare all concerned for a long siege. Our patient has been ill for some nine months already. As two years is practically an extreme limit for its duration, we can pretty confidently predict that another six months, or slightly more at the most, will see the end. That is, we are in for a siege of several months rather than one of a few weeks. And on top of this distressing information we have to add a climax of hopelessness. It is about as insidiously fatal as any disease can be. Hemsted has reported one case of apparent recovery, and Libman, whose experience in this disease is very wide, says that he has seen 3 cases recover out of a total of over 250 which he has observed. But certainly recoveries are so rare that we must consider the disease as practically uniformly fatal. One may hold out a faint ray of hope that an exceedingly rare case has not ended fatally. All of these facts must be imparted to some responsible members of the family, but I think there is only rarely justification for taking away all hope from the patient. They are generally satisfied with the information that there is a bacterial infection of the heart valves, that the fight is to get rid of these bacteria, and that it generally takes a long time. Everyone then settles down to the long fight, and the disease is so insidiously progressive that the patient scarcely realizes the fact that he is loosing ground slowly but surely.

You must remember to remind the family of the possibility

of the various complications which are apt to occur, such as the various types of emboli, especially cerebral, eventual cardiac decompensation, progressive anemia, pneumonia, and possible tuberculosis

In regard to the so-called bacteria-free cases or healed cases I can only refer you to Dr Emanuel Libman's articles. It is a most interesting aspect of the disease about which he knows more than anyone else.

Treatment includes all of the measures known to build up the patient's general condition, and, in addition, there have been used various measures of serum and vaccine therapy and transfusions. As I shall hope to show you this patient again or at least tell you the outcome of the case, we shall take these up at that time.

Subsequent Course — You will remember that early in the fall I showed you a case of non-hemolytic streptococcus endocarditis (A brief summary of the case was here given.) This patient died during the middle of March, a little over five months after you saw her, or, as nearly as we can tell, about fourteen months after the onset. This is just about what one expects in these cases.

Shortly after you saw her petechiae were discovered, as we predicted. Although never very abundant, some were usually present. She also had frequent tender fingers and toes and transient joint and muscle pains. There was an embolus to the tip of her right ear and rather numerous ones to her spleen and kidneys.

On January 10th there was a sudden complete left hemiplegia, from the effects of which she never completely recovered.

The temperature was irregular between 99° and 102° F., with occasional shoots to 104° F. The pulse was correspondingly elevated. There were frequent night-sweats, but chills were not a prominent feature. For some time the appetite was fair and then failed almost entirely. Loss of weight was progressive. Anemia was overcome and controlled for a long time by transfusions, but then became progressively marked.

Her heart never increased appreciably in size and she never suffered from any real decompensation. She developed numer-

ous extrasystoles and several changes in the quality of her systolic murmur, but no new murmur appeared

On February 20th she left the hospital for her home and, without the appearance of any new complications, flickered out like a candle about one month later. It is quite striking that during this month she had no fever.

The autopsy revealed the usual typical picture of the disease. I have not time to go into it at this moment, for I wish to emphasize the treatment.

The patient was most intelligent. She never lost hope, and thus we had the best possible co-operation. Every possible supporting measure was used to keep her generally in the best condition.

For a time she had autogenous vaccines. They did absolutely no good, and were only used because the family wished it. Sensitized vaccines were not used. They do no more good than the others in this condition. We might just as well realize and accept the fact that in non hemolytic streptococcus endocarditis vaccines, in large doses or small, at long intervals or short, sensitized or unsensitized, do absolutely no good.

Transfusions were used repeatedly. The blood was obtained from two different donors, one of whom was vaccinated with small doses of her organism, the other with large doses. As far as could be determined, there was no difference in the effect on the patient. In all she received eleven transfusions with a total of about 2600 c.c. of blood. Now, as I have said, the transfusions controlled the anemia for a long time and in this way undoubtedly prolonged her life. This we have seen repeatedly in these cases where the transfusions were not confused with vaccines. As anemia plays quite a considerable part in the downward path we may accept transfusions as an important therapeutic measure in these cases. We feel that they are valuable, but not as cures.

These transfusions from vaccinated donors were futile in sterilizing the blood-stream. This corresponds with our experience in the past and with that of others. To be sure, there was frequently a slight reduction in the number of colonies following

a transfusion, but this we see with straight transfusions and even after saline infusions. Here the number of colonies per cubic centimeter when treatment was started was 119. During its course the lowest number recorded was 63. So that in this case, as with others, all experience is against this procedure.

As this method was a failure, we tried large doses of serum from a vaccinated pony. In one week she received 700 c.c. of this serum. The colonies diminished to 41, but four days later were at the maximum for her—295. So that we accomplished nothing but a moderately severe serum sickness. In this connection it is of much interest to note what Dr. Cole of the Rockefeller Hospital has told me. He says that occasionally a horse highly immune to the pneumococcus will get up a pneumococcus endocarditis.

Our present-day methods against this disease are powerless. It is well to recognize this and to have the courage to give up repeating the same old useless procedures.

Before closing this case I want to give you a list of the more important references. It is most important for you to file them away for future reference. You must read these articles to round out your knowledge of the disease. In them you will find all the other references. If you have not yet started a proper system of medical filing, get busy at once, for you can do nothing in medical practice, research, or teaching without such a system.

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CLINICAL TALK BY DR LEO BUERGER
MT SINAI HOSPITAL

~~SYMPOSIUM~~
CYSTITIS A DISCUSSION CONCERNING ITS ~~ETIOLOGY~~

Inadequacy of Prevailing Methods in Arriving ~~at~~ ~~the~~ ~~exact~~ ~~diagnosis~~
of Cystitis Importance of Cystoscopic and ~~other~~ ~~examinations~~ ~~in~~ ~~correctly~~ ~~interpreting~~ ~~the~~ ~~pathologic~~ ~~changes~~ ~~due~~ ~~to~~ ~~this~~ ~~condition~~. Illustrative Cases

of cases which are now indiscriminately labeled as "cystitis," presumably masquerade as cystitis, but, in reality, suffer from a multitude of other lesions.

Were this exposition to do nothing else but emphasize that an attempt to make a diagnosis of cystitis from symptomatology and from certain easily ascertainable objective findings—such as urinary findings—is not only futile but fallacious, it will have accomplished its purpose. Let us pause for a moment to recount the case of a young girl who recently consulted me, and who had been treated for many weeks by her physician as a case of acute cystitis with the usual bladder irrigations, who had been becoming progressively worse in spite of the treatment—for here we have a most illuminating example of the worthlessness of clinical signs even when coupled with laboratory examinations, in the diagnosis of so-called cystitis.

CASE I.—Foreign Body, Encrusted (Petrified) Catheter in Contracted, Intensely Inflamed, Ulcerated Bladder of Female Giving Symptoms of Cystitis

A A, female, consulted me December 30, 1918, with a story that she had been in perfect health until about three months ago, when her bladder symptoms began. Since then she has complained of great frequency of urination, intense pain during the act, the symptoms becoming daily more and more severe, so that now she must void every ten or fifteen minutes during the day, and gets up at least three or four times during the night. She had been receiving daily irrigations of the bladder for more than six weeks without noticing any improvement whatsoever.

Cystoscopy, December 30, 1919, revealed an intensely inflamed, very much contracted, irritable bladder, the site of an intense cystitis, harboring a foreign body that gave the appearance of a coiled-up bougie or catheter of about No. 18 French, almost completely encrusted with lime or phosphatic salts.

On January 10, 1919, at operative cystoscopy done under anesthesia, with the author's operating cystoscope inserted into the bladder, the encrusted catheter was easily brought into view, the grasping or punch forceps of the operating cystoscope introduced through the special telescope made to seize the catheter, and the foreign body easily removed in the well-recognized, typical fashion, allowing the cystoscope to emerge first, the catheter in the grasp of the forceps following (Fig. 206).

The clinical lesson to be drawn from cases of this sort—and such errors in diagnosis occur not infrequently—may be summed up as follows. The symptoms of cystitis are not to be depended upon in diagnosis, for they give us no true knowledge as to the real lesion responsible for the clinical manifestations, the cystitis being but one of the products or complications, as it were, of a more profound or more serious affection, and finally, that treatment based upon symptomatic diagnostic conclusions can be of



Fig. 206.—Encrusted catheter removed through operating cystoscope in case treated for cystitis.

no avail. In fact, it may be well to emphasize right here at the beginning that the symptoms of cystitis do not at any time warrant the diagnosis of this affection any more than the recognition of sequelæ or complications of any other disease will explain the nature of the original malady. It is only with the application of the cystoscope, aided at times with the x-ray examination, that the correct interpretation of the existing pathologic conditions is possible.

The urologist who not infrequently encounters cases with su

pravesical lesions, when the mere clinical signs suggest the presence of a cystitis, has learned to apply the cystoscope, and no longer relies upon his examinations of urine and what the history may reveal. Not infrequently will an inflammatory lesion of the kidney with or without renal lithiasis masquerade under the guise of so-called "cystitis." The following case will illustrate

CASE II.—Infected Hydronephrosis, Multiple Renal Calculi, Ureteral Stricture at the Ureteropelvic Junction, Marked Perinephritis, Being the Lesions Discovered at the Time of Nephrectomy in a Patient who Consulted Me for Urinary Frequency and So-called Cystitis

R. S., female, May 23, 1914, gave a history of having bladder trouble, diagnosticated as inflammation. Recently she had another attack, voiding every fifteen minutes. Symptoms referable to the kidneys had been absent, except on one occasion some indefinite pain across the back.

Cystoscopy May 14, 1914, revealed evidences of cystitis, but disclosed that the chief lesion was in the right kidney. Catheterized specimens from the right kidney showed the presence of pus-cells and some red blood-cells, and markedly diminished functional capacity of that kidney.

α -Ray examination showed a calculus about $\frac{3}{4}$ inch long and $\frac{1}{2}$ inch wide, and several smaller calculi in the lower pole of the right kidney, and another stone presumably at the ureteropelvic junction measuring $1\frac{1}{2}$ inches in length by $\frac{1}{2}$ inch in width.

May 29, 1914, typical nephrectomy revealed an infected hydronephrotic kidney harboring the calculi described in the α -ray report, with marked dilatation both of the extra- and intra renal pelvis, an enormous hilus lipoma, marked perirenal inflammation, the calculi being situated in the pelvis and calyces.

At the ureteropelvic junction a stricture, probably of inflammatory nature, was observed, and in the cortex near the upper pole there was a small mixed tumor measuring less than $\frac{3}{4}$ inch in diameter.

Clinical Course—Almost immediately after the removal of the offending organ there was complete cessation of the symptoms of cystitis.

Symptoms of cystitis, therefore, may be the expression either of the reflex action of a diseased kidney with calculi, or the result of bladder lesions produced by the action of infected urine secreted by a diseased kidney.

The symptoms of cystitis may be the only ones, or at least the complaint which prompts the patient to consult the physician or the surgeon, even though a most extensive inflammatory lesion of the kidney, with or without calculus, may be present, for the latter may give no symptoms until the onset of extensive suppurative renal infarction brings about fever and chills.

CASE III.—*Cystitis Symptoms Predominating in a Case of Pyelonephritis, Infected Hydronephrosis, Calculus in the Upper Ureter, Perinephritis, Stricture of the Lower Ureter Associated with Cystitis, Complicated Later by Multiple Cortical Abscesses, Suppurative Renal Infarction Requiring Nephrectomy*

M S female, consulted me on the 29th of October 1918, because of urinary frequency, urgency, nocturia (three times at night), dysuria. She had been operated upon six months previously for a uterine condition, and the uterus was said to have been removed. It was only after careful questioning that she admitted having had intermittent lumbar pain, but this was not definitely located, and did not seem to annoy her sufficiently to be worthy of note.

Cystoscopy demonstrated an inflamed bladder (cystitis). The specimens from the left kidney were negative, but an obstruction was encountered at a point 8 cm removed from the right ureteral orifice. No urine was obtained from the right kidney, and because of the marked pyuria it seemed more than likely that the pus content of the urine was attributable to an infection of the right kidney.

X Ray examination October 31, 1918, showed a shadow opposite the third lumbar vertebra, about $\frac{1}{2}$ inch in length, presumably at the hilus of a somewhat enlarged right renal shadow. Although these findings were not very definite, the shadow was sufficiently distinct to warrant the suspicion that a renal or pelvic stone was present.

It was decided, therefore, that it would be advisable because of the pyuria and the obstruction in the right ureter to explore the right kidney, the diagnosis of hydronephrosis being made in view of the large amount of pus that was present in the bladder. This advice the patient refused to follow, and she passed out of my hands until December 1 1918, when she consulted me in great distress having had an attack of fever and exquisite lumbar pain so severe that she said "she could stand it no longer."

The patient was referred to the hospital, and on the 8th of

December her temperature rose to 105° F., and there was a distinctly palpable mass in the right loin

December 9th nephrectomy was done

Pathologist's Report — *Kidney Specimen* — The kidney is more than twice the normal size, capsule very much thickened



Fig 207.—Hydronephrotic kidney, showing two large foci of suppurative infarction which infiltrate the parenchyma

and edematous, there being a considerable increase in the fatty tissue about the kidney and renal hilus. When the kidney is opened a large amount of purulent urine is evacuated. The pelvis is very much distended, and on section (Fig 207) two large foci of suppurative infarction can be seen to infiltrate the paren-

chyma, causing a prominence at the external border of the kidney (Fig. 207), this on surface section showing numerous areas of purulent infiltration. In the capsule numerous milary ab-

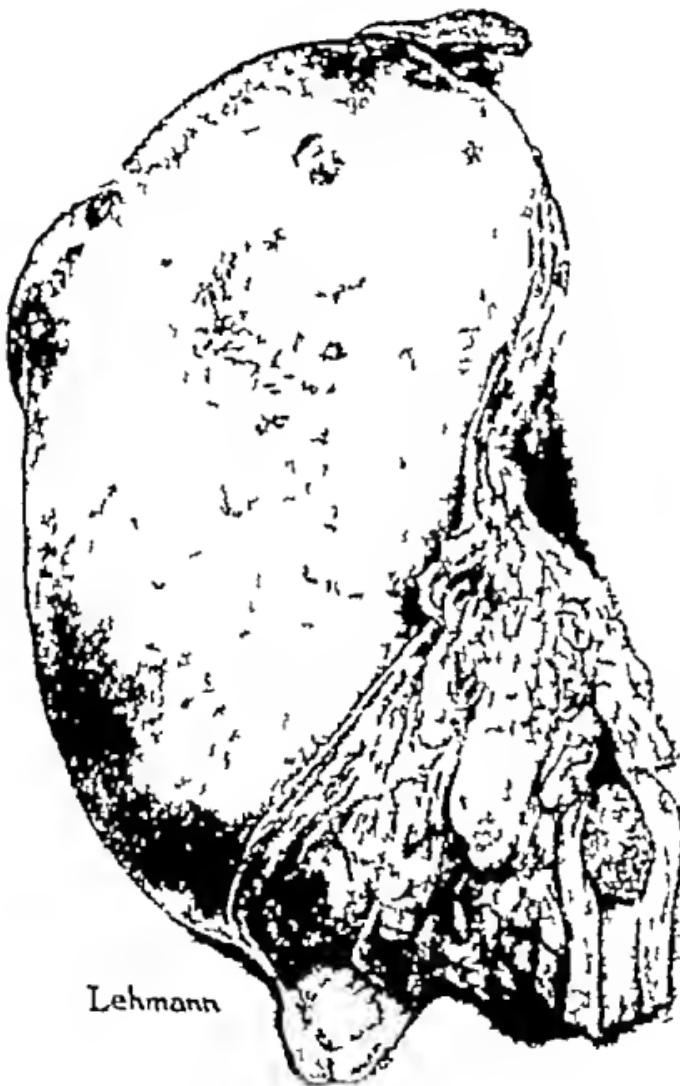


Fig. 208.—Same case as Fig. 207 depicting prominence at external border of kidney and ureteral calculus.

cesses are seen on the surface of the organ, and at the upper pole there is another focus of suppurative infarction.

In the ureter, just within a few centimeters of the pelvis, there

is a calculus about $\frac{1}{2}$ inch in length, embedded in swollen, velvety red, purplish mucous membrane, the ureteral wall being thickened to about 3 to 5 mm in this neighborhood (Fig 208)

Here was a woman operated upon for uterine trouble with a stricture low down in the ureter, suspected as being the result of inclusion of the ureter in a ligature, who had a shadow in the upper ureter region, near the pelvis of the kidney, hazy, it is true, but nevertheless somewhat suggestive, and who sought relief particularly because of bladder symptoms, *frequency of micturition, urgency, and pain on urination*. On palpation we found a somewhat enlarged kidney, and on cystoscopic examination an obstruction at 8 cm in the right ureter.

Clinically interesting was this case because of the fact that *frequency of urination predominated in the picture* until the occurrence of suppurative infarction in the kidney.

Clinical Conclusions—Cystitis, therefore, as suggested by the above striking illustrative examples may be merely a complication or concomitant resulting irritative local vesical alteration, brought about by an infection of one or more portions of the supravesical urinary tracts.

Vesical calculi, too, associated with infection of one or both kidneys or infection combined with renal lithiasis may give clinical pictures that may readily be confused with those of so-called cystitis. Just as foreign bodies of the bladder and other lesions to which we shall later refer may clinically simulate those manifestations of deranged micturition usually incorrectly set down by the practitioner as "cystitis," so also large vesical calculi may be completely overlooked unless the methods of cystoscopy and x-ray examination be applied.

Before occupying ourselves with a detailed discussion of some of the many pathologic lesions that may be overlooked when a diagnosis of cystitis is made on clinical symptoms alone, let us cite one very striking example of the insidious nature of renal and vesical lesions as far as localizing symptoms are concerned.

CASE IV—*A man with the symptoms of chronic cystitis develops a most extensive infection of one kidney, with marked perinephritis,*

multiple abscesses of the kidney, without at any time presenting any manifestations of pain or lumbar tenderness in the region of the affected kidney, although bladder symptoms have been present for a long time, and some of the toxic symptoms and effects of absorption had been developed a short time before nephrectomy was carried out

M. N., male, consulted me December 28, 1917, the history being that of *protracted cystitis*, possibly associated with stricture

X Ray examination revealed a vesical calculus, which on some of the plates had a spheric outline $2\frac{1}{2}$ inches in diameter in others had an elliptical shape $2\frac{1}{2} \times 1\frac{1}{2}$ inches in diameter

Suprapubic cystotomy was carried out on January 10, 1918, and the calculus removed, the patient making an uneventful recovery, except for one attack just before his discharge from the hospital, which afterward was diagnosticated as a possible evidence of pyelonephritis

The suprapubic wound healed promptly after the removal of the drainage-tubes, and the patient left the hospital with practically no disturbances of micturition, but showing the distinct signs of severe attacks of pyelonephritis. He did not appear strong and healthy, and recurrences of attacks of pyelonephritis were considered extremely likely, and, in fact, were to be expected.

The patient was again admitted to the Mt. Sinai Hospital on February 27th, with a temperature of 101° F., no renal tenderness, a haggard look, and urine containing a fairly large amount of pus. It was again a question as to whether the temperature was due to the infection of the left kidney without local manifestations, or whether some intercurrent or other infection, such as tuberculosis, was present.

On February 28, 1918, another cystoscopic examination was performed to determine whether we were dealing with retention of purulent urine in the kidney or not. At this time it was easy to obtain a perfectly good view of the bladder, because the stone had been removed, and the mucous membrane was found almost normal except for some congestion. It was with surprise that *two ureters were discovered* on the left side, the upper or posterior one doubtlessly having been covered up by the large calculus at our previous cystoscopic examination. All three ureters were catheterized, specimens being obtained from the right kidney, and from the upper or posterior and lower or anterior ureteral orifices on the left side.

	Right.	Left upper	Left lower
Reaction	Acid	Acid	Acid
Albumin	Trace	Trace	Trace
Urea	1 1 per cent	1 4 per cent	0 7 per cent
Microscopic	Few epithelial cells Few red blood cells No pus-cells	Few epithelial cells Few red blood cells No pus cells	Few epithelial cells Few red blood cells Many pus-cells
Culture	Few hyaline casts Sterile	Few hyaline casts Bacillus coli communior	Few hyaline casts Bacillus coli communior

To summarize, we have a fairly normal functioning right kidney with urine containing a trace of albumin, a left kidney containing two pelvis, the lower pelvis corresponding to the upper ureter, presumably normal except for the presence of *Bacillus coli communior* in culture, and an upper pelvis evidently infected with urine containing many pus-cells, diminished urea output, and infected with *Bacillus coli communior*.

Thus, we were dealing with a man who had had recurrent attacks of pyelonephritis in a kidney with a double pelvis, the lower portion of which was evidently healthy, the other or upper portion harboring one or two calculi, and the seat of an extensive pyelonephritis.

Diagnostic Conclusions — In a man who had had a large vesical calculus, and in whom the γ -ray findings indicated the presence of a fair-sized calculus in the left kidney, the clinical course as well as the cystoscopic findings pointed to severe infection of at least the upper half of the left kidney, the lower pelvis being uninvolved. The insidious nature of the process as evidenced by the absence of local pain, renal colic, or local tenderness, the presence of attacks of fever with general symptoms rather than localizing phenomena, made the interpretation of the clinical course and the surgical indications somewhat difficult. However, from a consideration of the findings, it was possible to make the following deduction regarding the anatomic lesions present.

Thus, we surmised that the upper portion of the kidney was the seat of an inflammatory process with multiple abscesses, or at least extensive pyelonephritis, and suppurative areas in the

parenchyma, and furthermore, because of the long history, and the absence of a true pyonephrosis (as indicated by cystoscopic



Fig 209.—Infected kidney with double pelvis. Deposition of inflamed fat about the upper two-thirds of the kidney enlarged upper ureter normal lower ureter normal lower pole with cut surface through the isthmus

findings), and the absence of pain and tenderness, we were in all probability dealing with an inflamed kidney, whose exterior was

enveloped in markedly thickened perirenal adipose tissue, that is, a kidney surrounded by enormous fat production, and enveloped as it were or encased in a mass of inflammatory tissue. The operative findings corroborated these diagnostic inferences.

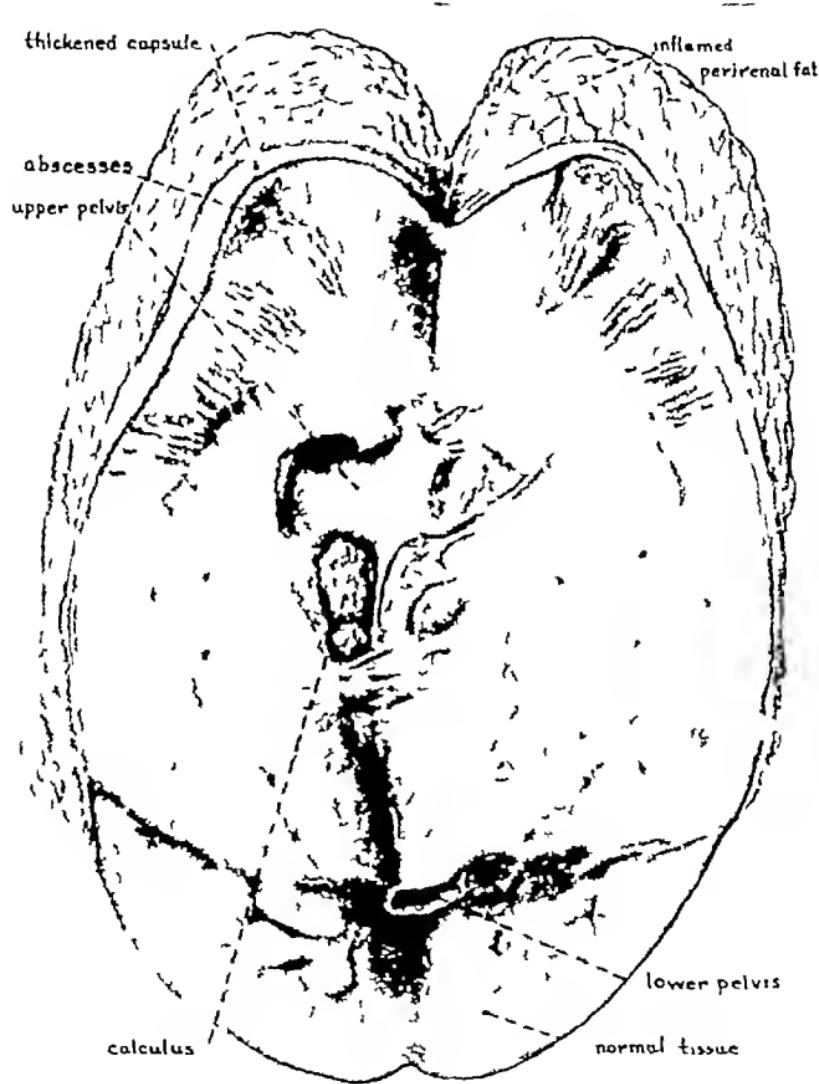


Fig. 210.—Kidney bisected, showing perirenal inflammatory fat, two stones in upper pelvis, with multiple abscesses, inflamed edematous upper three fourths of parenchyma and normal lower pole.

Operation, March 5, 1918, typical nephrectomy through the usual Albaran incision, with the removal of an infected kidney.

provided with two pelvis and fused by its lower pole with the sister organ of the other side (Figs. 209, 210)

Is it permissible, then, for the practitioner to make a diagnosis of cystitis? In a general way it may be suggested that a tentative diagnosis of cystitis may be made without danger of falling into error, particularly in those cases which present acute symptoms of but short duration, one week or two at the most, and in which a complete recovery follows as far as all subjective manifestations and urinary changes is concerned. But even in such instances, as will be shown later on, further investigation of the character of the bladder interior and the condition of the kidneys and ureteral tract, supplemented by the x ray is advisable, lest lesions of great import be overlooked. *In the chronic cases a purely clinical diagnosis of cystitis is in the present state of our knowledge of urology fraught with grave danger and may jeopardize the health of the patient, as well as be responsible for the destruction of a kidney that might otherwise be saved.*

Before entering into a detailed discussion of just how far our clinical judgment in the recognition of cases of so-called cystitis may be allowed to venture, let us pass in review some of the many pathologic conditions that may offer a clinical syndrome indistinguishable from the text book picture of cystitis, and that may be grouped according to the site of the process, as follows:

LESIONS WITH SYMPTOMS SIMULATING CYSTITIS

RENAL LESIONS

- 1 Tuberculosis of the kidney (very common)
- 2 Calculi with or without infection.
- 3 Hydronephrosis
- 4 Urinary concretions
- 5 Renal crises with bladder symptoms
- 6 Pyelitis
- 7 Pyelonephritis
- 8 Pyonephrosis
- 9 Metastatic or embolic infectious lesions

URETERAL LESIONS

1 Ureteral calculi

BLADDER LESIONS

- 1 Tuberculosis
- 2 Ulcers
- 3 Foreign bodies
- 4 Calculi and concretions
- 5 Tumors of the bladder, carcinomata, papillomata
- 6 Leukoplakia
- 7 Malakoplakia
- 8 Syphilis
- 9 Edematous lesions about the sphincter, being an extension of lesions in the urethra and prostate
- 10 Intravesical intrusion of prostatic adenomata (Albaran lobe)
- 11 Diverticula

LESIONS OF THE BLADDER NECK

- 1 Fibrosis of the sphincteric region
- 2 Contracture of the neck of the bladder (including so-called median bar)
- 3 Submucous adenomata and fibromata

PROSTATIC LESIONS

- 1 Prostatitis
- 2 Prostatic congestion
- 3 So-called hypertrophy or fibro-adenoma, or adenoma
- 4 Fibrosis
- 5 Tuberculosis
- 6 Carcinoma
- 7 Prostatic abscess
- 8 Rare prostatic tumors (sarcoma)
- 9 Prostatic calculi

LESIONS OF THE SEMINAL VESICLES

1 Vesiculitis and perivesiculitis.

2. Tuberculosis

URETHRAL LESIONS

1 Gonorrhreal urethritis

2 Stricture of the urethra.

3 Calculus in the posterior urethra, particularly behind the verumontanum

4. Malformation and urethral ureter

In addition to this large array of pathologic processes that may simulate and give symptoms of cystitis, there are causes outside of the bladder (extravesical) that may be responsible for similar manifestations. They are as follows

EXTRAVESICAL LESIONS

- 1 Pelvic abscess (infection from the adnexa and uterus)
- 2 Pelvic abscess from appendicitis
- 3 Tumors of the uterus and adnexa
- 4 Hemorrhoids and carcinoma of the rectum
- 5 Vesicovaginal fistulae
- 6 Vesico-intestinal fistulae
- 7 Penicystitis, etc.

In addition to these may be mentioned a number of other general conditions that may present pollakiuria and even urgency, and that are often mistaken for cystitis. Thus, may be mentioned—

- 1 Constipation
2. Onanism, with congestion of the sexual organs
- 3 Neuroses.
- 4 Chemical alterations of urine phosphaturia, etc.

CLINICAL LESSONS IN DIFFERENTIAL DIAGNOSIS

To enumerate and recite in detail case illustrations exemplifying erroneous interpretation of conditions indicated merely by

name in the above lists would take me far beyond the scope of this lecture. Let us rather, therefore, cite only a few instances in the groups mentioned here, where symptomatology has been deceptive, and other means for diagnosis had to be employed.

Renal Tuberculosis Simulating Cystitis—When a mother comes to you complaining of persistent bed wetting in the case of her child, although he or she has attained the age of six, eight, or ten years, and has perhaps noticed, too, that the child voids more frequently than it should—be this phenomenon unaccompanied by any other manifestations—the possibility of the existence of a tuberculous process in one kidney and in the bladder must be thoroughly entertained. *For frequency of urination is so often the initial symptom of a renal tuberculosis and mistaken for cystitis that the presence of this condition must ever be borne in mind.* So that, when a child who appears to be somewhat too old to continue to have *enuresis nocturna* comes to us for examination, a specimen of urine is to be carefully scrutinized for the presence of pus and tubercle bacilli, and we will not infrequently be surprised to find an enlarged kidney plus tubercle bacilli in the urine of a child who has been regarded as having either *enuresis nocturna* or *cystitis*.

The busy urologist is wont to see almost daily an individual whose bladder has been washed for weeks or months, whose urinary frequency, urgency, nocturia, painful micturition, and pyuria have been attributed to and treated for simple inflammation of the bladder. The stories of such patients offer a clinical lesson that is not only most illuminating in emphasizing the futility of basing diagnosis on the means at the disposal of the practitioner, but is also of sufficient importance to demonstrate the mistake of applying treatment before a positive diagnosis has been made. For it is because of the incorrect attitude of security and false hope of future cure instilled into the patient by the initiation of local methods of therapy that the patient is lulled into a mistaken feeling of confidence and anticipation of early relief from a distressing complaint which is certain to become worse as the days pass by, coupled with daily extension of tuberculous lesions in the bladder as well as in the kidneys, each

moment of procrastination decreasing the ultimate chances of permanent recovery.

Renal and Vesical Tuberculosis Without Tubercle Bacilli in the Urine.—It is because of the difficulty of finding tubercle bacilli in many cases of renal and vesical tuberculosis that even the urologist is apt to be mistaken, and may find it difficult to be certain as to the exact nature of the lesion he has to deal with. The application of the cystoscope, however, will demonstrate that one kidney is involved or suspicious of tuberculosis, and will lead the experienced urologic surgeon to make a tentative diagnosis of renal tuberculosis, even though shadows of calculi may be demonstrated in the x-ray, and even though these are distinctly seen within the renal shadow of a suspected kidney.

It is not only with the cystoscope that the diagnosis of renal tuberculosis can be made, but the practitioner has at his disposal a very simple method in which he should acquire experience, namely, that of detecting the existence of a tubercular ureter by palpation through the vagina.¹ Were a routine vaginal examination carefully made with a view to palpating a diseased ureter in all instances of suspected cystitis in married women, many errors could be avoided. Just as the differential diagnosis between chronic appendicitis in women and a ureteral calculus low down in the ureter, lies within the reach of the examining finger, so also the induration of a tuberculous ureter will become readily recognizable after some practice.

CASE V—Case Treated for Eight Months for Cystitis, with a Stone Shadow Within the Right Renal Area, an Indurated Ureter Felt per Vaginam, an Ulcerative Cystitis with Urine Negative for Tuberculosis, Proved to Have a Tuberculous Process by the Removal of a Tuberculous Kidney Harboring Stone

H. S., female, had been troubled with urinary frequency for about eight months, there being a burning pain during the act, bladder irrigations having been recommended by her physician for "cystitis."

Cystoscopy, March 11, 1918, revealed an intense cystitis.

¹In the male, rectal examination will often disclose data of value either in the form of tuberculous vesicles or prostate, and more rarely a thickened ureter may be felt.

with superficial purulent exudate over superficial erosions or ulcerations, an obstruction in the right ureter at 4 cm above the right ureteral orifice, an absence of flow from the right kidney, the specimens from the left kidney being negative and showing good renal function. The bladder specimens showed pus, trace of albumin, no sugar, numerous *streptococci* and *bacilli in spreads*, *tubercle bacilli being absent*.

α -Ray examination disclosed two shadows within the right renal area, each about 1 cm in diameter, between the second and third lumbar transverse processes, doubtlessly indicative of the presence of calculi. The renal outline was suggestive of a large organ. In the pelvic region at a point corresponding to the site of the ureteral obstruction there was a suggestive shadow, possibly a calculus or calcification in the ureter.

Vaginal examination disclosed a hard ureter, an indurated mass, which could have been mistaken for peri-ureteritis, or could have been regarded as an inflamed ureter of the tuberculous variety.

Tentative Diagnosis — In brief, in a case with vesical symptoms, possibly right ureteral colic, with urine containing no tubercle bacilli, but pus, whose right ureter was obstructed at a point 4 cm from the vesical orifice, whose left kidney was normal and whose α -ray plates showed two stones in the right kidney, and a possible small stone in the right ureter, the question arose as to whether we were dealing with a case of calculous disease alone, with obliteration of the right ureter due to calculus in the ureter and consequent inflammation, or whether we were dealing with a combination of renal tuberculosis and renal calculi. Was it expedient to go down on the right ureter with the view to removing the calculus suggested by the shadow in the plate, or was it preferable to go in on the kidney which per abdomen could be palpated as distinctly enlarged, and which doubtless harbored at least two calculi?

In view of the absence of tubercle bacilli a positive diagnosis could not be made, but because of the ureteral obstruction, the uncertainty of the nature of the ureteral shadow, because of the presence of the hard cord felt per vaginam, and because of the feeling that the removal of the ureteral calculus in such a hard ureter could not be followed by good functional results, it was decided to expose the kidney, which I proceeded to do at the New York Polytechnic Hospital and Medical School March 14, 1918.

March 14, 1918, nephrectomy through an Albaran incision revealed a very much enlarged tuberculous kidney, and a ureter

about the thickness of a man's thumb was liberated and cut through with some difficulty. The kidney was removed in the usual fashion and the specimen revealed the following (Fig 211).

The kidney was almost twice the normal size, surrounded by an enormous amount of perinephritic inflammatory tissue. On section it showed a distinctly tuberculous kidney, in the pelvis

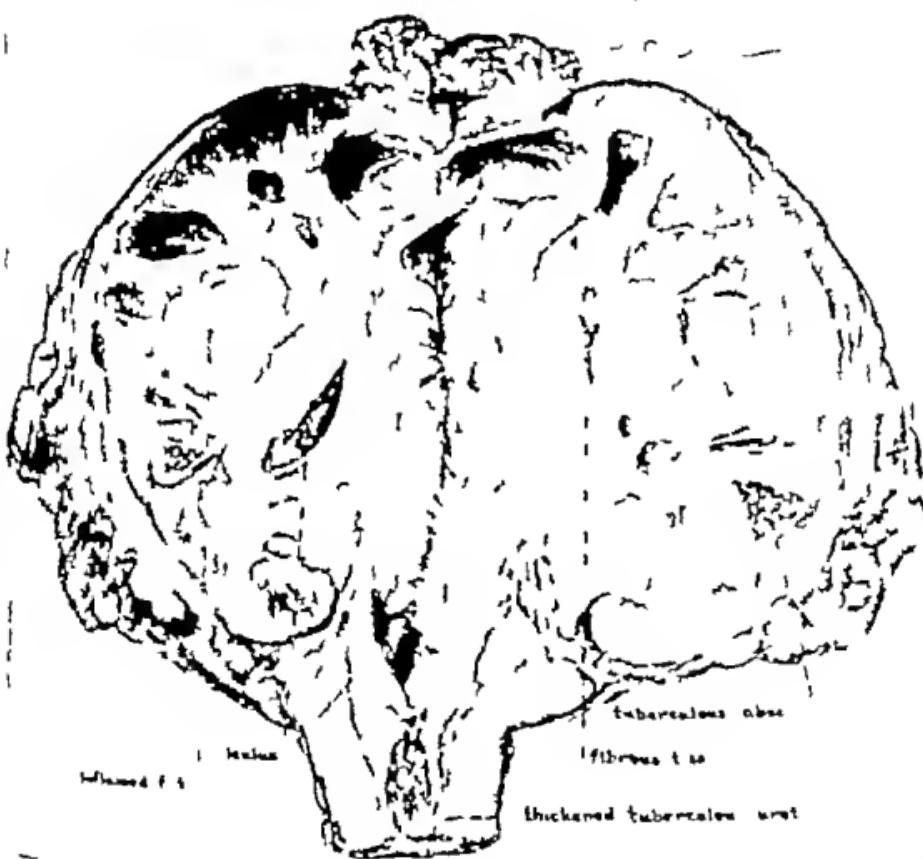


Fig 211.—Tuberculous kidney with calculi in a case diagnosed as cystitis.

of which two calculi were lodged. Interesting was the very large amount of fat formation in and about the pelvis and in the hilus, the dense masses of connective tissue about the pelvis, and the cavities, some of which were tuberculous and some due to the secondary dilatation produced by the presence of the calculi. The ureter showed the typical tuberculous lesions, its walls being enormously thickened (Fig 211).

Conclusions—We have here an illuminating example demonstrating, first, the wisdom of exposing rather the kidney than the ureter when we are in doubt regarding the presence of a tuberculous lesion, and one proving, second, that unless a shadow in the lower ureter be definite, the presence of an indurated cord per vaginam must be regarded rather as an evidence of tuberculous

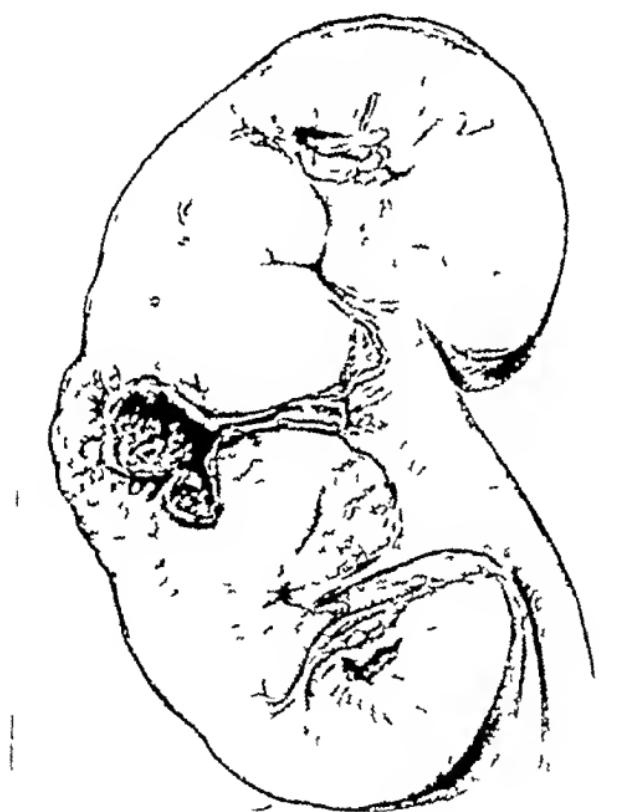


Fig. 212.—Tuberculous kidney, showing development of tuberculous cavity and miliary tubercles in a case treated for cystitis

ureteritis than of the presence of a calculus unless the outline of such calculus be distinctly felt, and third, indicating again the *error of entertaining a diagnosis of cystitis when the true nature of the lesions can only be ascertained by special urologic methods*

Tuberculosis of the Kidney Diagnosticated as Cystitis and Treated as Such—Cases of this sort are so numerous in my own

records that it would be a work of supererogation to do other than merely cite a few striking examples

CASE VI—Tuberculosis of the Kidney with Symptoms of Cystitis, Diagnosticated as Such and Treated as Such for Six Months, Operated Upon for Uterine Trouble, in Order to Cure the Cystitis

L R, female, consulted me August 27, 1917. She had had increased frequency of urination for about six months and had been passing urine every ten to fifteen minutes during the day as well as at night. Three months previously she had been operated upon at a hospital in the city, with the diagnosis of cystitis, the latter having been regarded as due to pressure of her uterus. No relief of the symptoms after operation was obtained, the pain on voiding and the frequency persisting and being intense and becoming more severe.

Cystoscopic examination made the diagnosis of right renal tuberculosis a positive one.

The kidney specimen obtained by nephrectomy (Fig. 212) showed an organ almost normal in external appearance, except that over the middle of its convex border there was a large area, over whose surface there were numerous solitary and agminated tubercles. Corresponding to this on section, as well as illustrated in the figure, there was a tuberculous cavity with numerous tubercles in the neighborhood.

CASE VII—Renal Tuberculosis Giving the Symptoms of Cystitis Only, in a Case Treated with Bladder Irrigations for More than Six Months, the Kidney Showing Extensive Cavernous Tuberculous Lesions

M U, female, had been complaining of painful and frequent urination for six months, for which she was treated with bladder irrigations, the diagnosis of cystitis having been made.

Cystoscopic examination made on May 21, 1918, made the diagnosis of left renal tuberculosis easy, the usual lesions of tuberculosis being present in the bladder, the specimens from the left kidney containing pus and tubercle bacilli.

Nephrectomy on June 1, 1918, revealed an enormous hydro-ureter due to stenosis of the lower end of the ureter by reason of tuberculous lesions, and the kidney seen in Fig. 213 of the typical cavernous tuberculous type containing large cavities lined with cheesy tuberculous material.

This case illustrates beautifully how extensive the lesions in the kidney may become while the patient is being treated for "cystitis."

Gonorrhreal Infection of the Ureter and Kidney Pelvis Simulating Cystitis—The fallacy of treating cases for cystitis, when

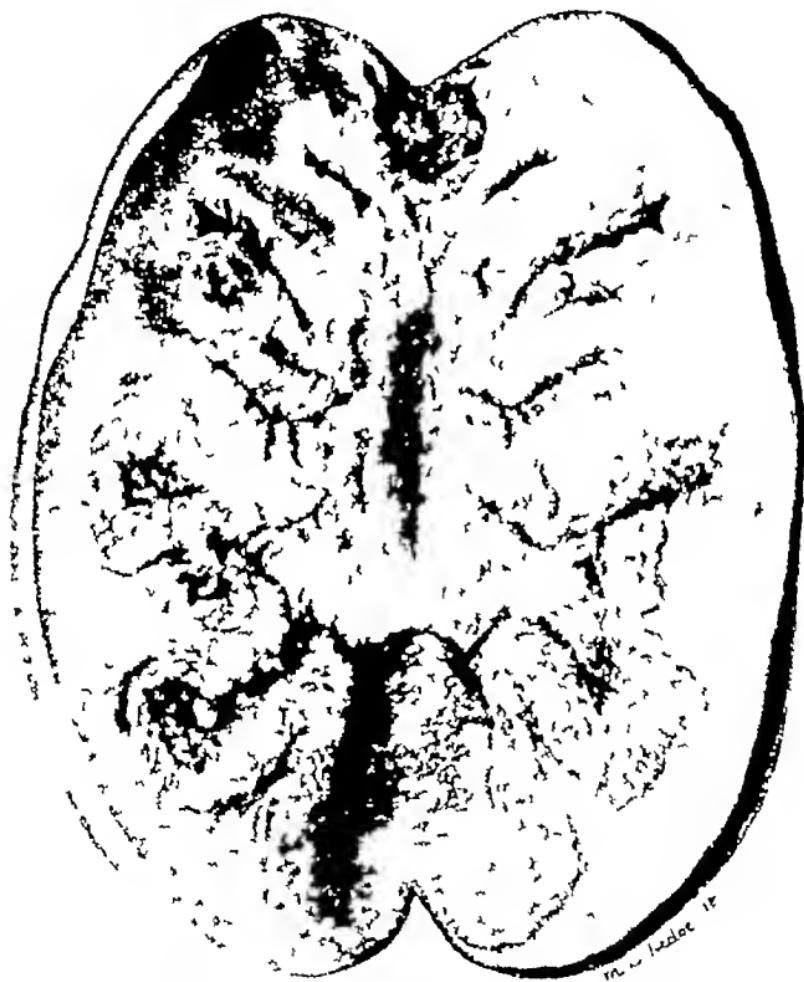


Fig. 213.—Tuberculosis of the kidney with extensive cheesy degeneration and cavity formation in case diagnosed as cystitis

we may be dealing with an infection of the whole urinary tract perpetuated by a focus of gonorrhreal infection in the ureter or pelvis of one or both kidneys, is well illustrated by the following case, which is not at all unique in this sense

the five specimens collected from the left kidney, a fact which practically rules out contamination with the catheter. Positive findings are also reported in the specimens obtained from the bladder. The presence of pus-cells in the left specimens further corroborated the diagnosis of infection of the ureter and pelvis of the kidney with the gonococcus. An x-ray examination on January 8th was negative. On January 13th cystoscopy was again done, and the pelvis of the kidney and ureter were washed out with 15 c.c. of a 20 per cent argyrol solution, about 5 c.c. being allowed to remain in the renal pelvis, the pelvis and ureter being irrigated with the rest of the solution.

In short, a case of undoubtedly gonorrhreal infection of the left ureter and left renal pelvis and bladder wall, the patient having been given one treatment of the pelvis and ureter with 20 per cent argyrol. The improvement was most remarkable after this treatment, so that a subsequent cystoscopy showed that the bladder was very much improved, the granular appearance having almost disappeared.

Within three weeks the patient was well.

Enough has been adduced in the way of clinical material to demonstrate how tuberculosis of the kidney *par excellence*, as well as renal calculi with or without infection, pyelitis, and pyelonephritis, may offer insufficient clinical evidence to warrant the diagnosis of a renal affection without the aid of cystoscopic and x-ray examination, and how bladder and so-called cystitis symptoms may dominate the clinical picture.

One other type of insidious encroachment upon the integrity of the renal organs by metastatic or embolic bacterial invasions may be worthy of brief mention.

Metastatic or Embolic Infectious Renal Lesions Without Renal Symptoms Mimicking the Clinical Picture of Cystitis

CASE IX.—W. E., running the gamut of multiple suppurative lesions from an unknown primary focus after a short period of bacterial invasion of the blood with *Staphylococcus aureus*, suddenly voided cloudy urine, accompanied with frequency, urgency, and the usual symptoms of cystitis. There were no symptoms referable to either kidney, no tenderness, no enlargement of these organs, but the pyuria persisted.

Cystoscopy demonstrated that the specimens from the right

kidney were freely admixed with pus, which on culture exhibited *Staphylococcus aureus*

Diagnosis—Embolic infection of the right kidney with *Staphylococcus aureus*, the foci communicating freely with the pelvis

Another and even more remarkable instance of the fleeting transitory course of certain types of embolic renal infections that complicate a *Staphylococcus aureus* lesion elsewhere in the body, with total absence of any symptoms referable to the kidney, was noted in one of our recent cases.

CASE X.—M. E. presented an indolent infection of the dorsum of the foot, accompanied with edema of the foot, and an enlarged inguinal node, and the usual evidences of lymphangitis extending from the foot to the knee. An incision over the abscess evacuated pus which could be traced upward for some 5 inches along the lymphatic channels, all of these being widely laid open and the wound packed.

Two days later there was a chill, and on the following day there was marked urinary frequency, hematuria, considerable pus in the urine, frequency and bloody urine existing for some two days, these symptoms subsided on the third day, when the urine became clear, only a few pus-cells being discernible in the specimens under the microscope.

Diagnosis—Metastatic, embolic suppurative focus rupturing in the pelvis, initiated by chills, a complication of a *staphylococcus* abscess on the dorsum of the foot, associated with lymphangitis, there being at no time any symptoms referable to the kidneys, bladder symptoms predominating.

It will not be necessary to dwell here upon the various ureteral lesions that may simulate cystitis, for these are, as a rule, not difficult to diagnosticate, particularly the presence of ureteral calculi.

Callosus Ulcer Simulating Cystitis

Among the bladder lesions, however, there is one which is overlooked when patients are not given the benefit of a cystoscopic examination, namely, that most distressing a callosus ulcer of the bladder. This is a lesion which may unrecognized for years, but which should be suspecte

cases of intractable cystitis occurring particularly in young women, where tuberculosis of the kidney or bladder can be excluded. In several such cases that have come under my observation a most remarkable and rapid cure was effected by the removal of the ulcer-bearing area with the punch forceps through the operating cystoscope. The history of one such case will be presented here.

CASE XI.—B. S. consulted me November 21, 1912, because of an intractable cystitis, severe pain on micturition, and ardor urinæ.

Her bladder symptoms began in November, 1911, when she noted that she had to void more frequently than ever before, and that the act of urination was accompanied with burning and pain. At first she paid little attention to these symptoms until they became so severe toward the end of her pregnancy that she sought the advice of a physician, who prescribed internal medication. After her confinement in May, 1912, the symptoms did not abate, but gradually became more severe, so that she had to urinate every hour or less during the day and frequently during the night. In August, 1912, about nine months after the onset of her illness, she sought the advice of Dr. K., who found that the urine contained a large amount of pus and some blood. She remained under treatment for almost four months, receiving frequent vesical irrigation with silver nitrate without improvement. The dysuria, painful micturition, and increased frequency persisted, so that finally she was compelled to void as often as every fifteen or thirty minutes during the day and every fifteen or thirty minutes during the night. At times the urine seemed to contain clots of blood. Lately the pain was so severe that she would "double up" during the act of micturition.

On November 21, 1912, cystoscopic examination was made. The whole trigone was markedly inflamed, the changes on the right side being more intense than on the left. The left ureter could be detected as a markedly swollen orifice embedded in edematous mucous membrane. Not far removed from the left orifice occupying the middle and right cornu of the trigone there was a large whitish polypoid mass of exudate, the surface of which presented areas of brownish and blackish discoloration, giving the impression of a tumor mass about the size of a hickory nut (Fig. 214). The right ureteral orifice could not be discerned, it being evidently hidden by the mass. Far back in the bas fond, situated somewhat to the right and the median line, there was a superficial defect in the mucous membrane, and not far removed

from this in the posterior wall another smaller abrasion. The rest of the bladder appeared to be pale and almost devoid of changes. The presumptive diagnosis after this examination was chronic ulcer involving the region of the right ureter and trigone, with intense trigonitis, cystitis with some superficial defect, and possibly right renal tuberculosis.

The urine contained a large amount of pus, numerous red blood-cells, and cultures showed *Staphylococcus albus*. No tubercle bacilli could be found.

On December 6th she was again examined (without general anesthesia), the vesical changes being very much the same as those noticed at the last examination. Clear urine was obtained from both kidneys, the right side excreting 17 per cent. urea, the centrifuged specimen containing a few epithelial cells and a few white blood-cells. The left ureteral specimens contained 2 per cent. urea, microscopic examination showing a very few red blood-



Fig. 214.—Callosus ulcer (2 cases)

cells. Both specimens contained a very faint trace of albumin. The cultures from both sides were sterile.

The findings at this cystoscopy, therefore, were the absence of renal involvement, the presence of a chronic ulcer in front of the right ureteral orifice, and cystitis with an area of erosion.

On December 8th a catheterized bladder specimen again showed numerous staphylococci (*Staphylococcus albus*) and an unidentified Gram positive bacillus. No tubercle bacilli were found. A twenty four hour specimen also proved negative for tubercle bacilli.

Excision of the Ulcer—With the diagnosis of chronic ulcer covered by a tumor like exudate now well established, and with the striking curative result obtained in a previous case in mind, it was determined to excise the whole ulcer area and the polyoid mass by means of the punch forceps through the operating cystoscope.

Therefore, on December 8th the whitish polyoid mass was

completely removed and the base of the ulcer punched out, both soft and hard pieces of tissue being obtained. The margins of the ulcer and a fairly large firm fibrous piece (undoubtedly representing the bottom of the ulcer) were identified both macroscopically and microscopically. All of these pieces were sent to the pathologic laboratory for histologic examination.

On the day following the procedure the patient already gave evidences of improvement as far as the urinary symptoms were concerned. Three days later the improvement was already marked, the patient being able to hold 10 ounces of urine without trouble. The urine obtained by catheter was almost clear, contained a few red blood-cells and a few white blood-cells.

On December 19th, ten days after the excision of the ulcer, cystoscopic examination showed a complete absence of the ulcer. The right ureter was now distinctly visible, the trigone just in front of it being occupied by several small ecchymoses. The mucous membrane was swollen and the ulcer evidently in a process of rapid repair. The catheterized specimen of urine was perfectly clear and the patient regarded herself "well," voiding only three times during the day and not at all during the night. There are absolutely no vesical symptoms.

Cystoscopy, January 10, 1913, revealed a practically normal bladder, there being no vestige of the ulcerated area. Pale mucous membrane in front of the right ureteral orifice was the sole indication of the former lesion. The patient considered herself "cured."

Cases of this sort are beautiful examples of the value of exact diagnosis in bringing about a cure of otherwise practical hopeless affection.

Another interesting lesion which is frequently overlooked is solitary superficial ulcer of the bladder. Upon close investigation of some of the cases that come to us suffering with terminal hematuria and cystitis there will be found a small *superficial ulcer* somewhere in the posterior wall or roof of the bladder. Such superficial ulcers do not require the radical treatment of excision except in rare instances. Some of them respond to mercury injections, others to the high-frequency treatment.

Lesions of the Bladder Neck—Because the fibrotic condition, the so-called contractures of the neck of the bladder and median bars, as well as fibroses of the prostate giving similar symptoms,

are so often confused in their clinical manifestations with "cystitis" it may be well to dwell for a moment upon this interesting pathologic change.

By the general term *contracture of the neck of the bladder* I wish to designate all those pathologic lesions involving the region of the internal sphincter and adjacent supramontane and posterior urethra, that result in the production of a greater or less coarctation of this portion of the urethral canal. The pure new growths, particularly adenomata, that may be located in the same territory, belong more properly to the class of cases known as hypertrophy of the prostate or adenomata of the prostate, and when they are unaccompanied by inflammatory or fibrotic lesions leading to a stenosis of the sphincteric region will not be regarded as belonging to the class of contracture of the neck of the bladder.

It was the pathologic study of cases that gave the clinical symptoms of contracture of the neck of the bladder, cases that did not belong to the class of adenomata that led me to adopt a more extensive operative procedure than those which merely have for their purpose the removal of a small portion of tissue from the floor of the sphincteric region as described by Young. These pathologic investigations on material obtained by a wide exsection of tissue from the affected region led me to the conclusion that, although no single lesion is characteristic for all cases and although a combination of lesions may occur to make up the complex of contracture of the neck of the bladder, nevertheless the anatomic alterations which produce the narrowing of the bladder outlet are so extensive and deep that a more radical surgical procedure should be followed by better results than some of the methods applied heretofore.

My own pathologic studies have brought to light that we are dealing with a combination of lesions in some cases, single lesions in others, always, however, representing invasion of the tissues situated at some distance from the mucous membrane side of the sphincter. These lesions may be a simple fibrosis showing no signs of previous inflammatory process, although such may have been present, fibrosis being superficial or deep, or there may be a fibrosis accompanied by an inflammatory process, or there may

be an inflammatory lesion involving the mucous side of the sphincteric region extending downward for a variable distance into the surrounding tissue or muscles, forming an inflammatory and fibrosclerotic sheath of varying thickness, or there may be a diffuse adenomatous invasion of the sphincteric region with or without periacinar inflammation, accompanied by more or less fibrosis. And finally, there are the mixed forms in which any

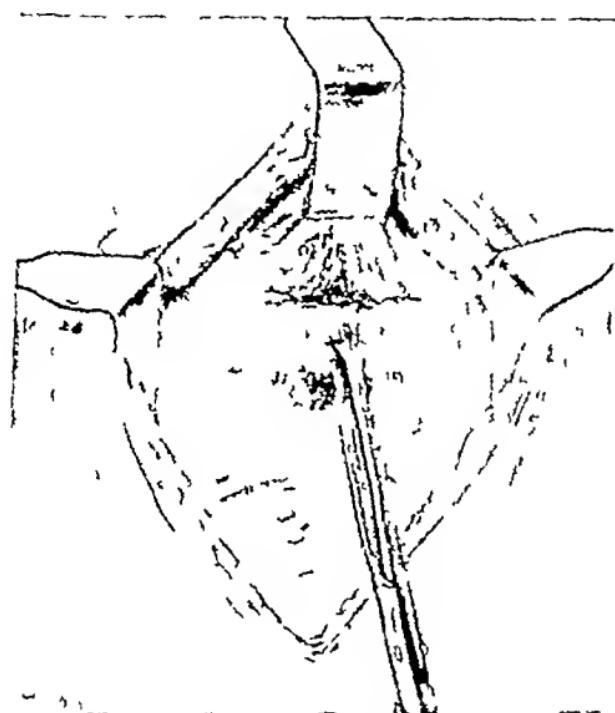


Fig. 215.—Region of the fibrotic sphincter (contracture of neck), showing tenaculum forceps elevating the floor of the sphincter before excision, according to the author's operative method

of the above changes may be accompanied by accidental formation of precocious adenomata or fibromata

Clinical Examples—When a young or middle-aged man comes to us with a story of frequency of urination, either with clear or cloudy urine, *who is said to have had cystitis*, or who may have been regarded as a neurasthenic if his urine be negative, let us refrain from making the diagnosis of either neurasthenia or cystitis until we have carefully ruled out the possible existence

of contracture of the neck of the bladder Too many young men are being daily designated as neurotic, and just as many cases with this same lesion are being regularly treated for cystitis.

Nocturia, diurnal increased frequency, urgency, irritative phenomena in the region of the perineum, indefinite and vague pains in the hypogastrium, particularly when the bladder is full, are the chief clinical symptoms As the disease progresses, however, secondary cystitis and not infrequently calculous formation, and even later infection of one or both kidneys with either pyelitis or pyelonephritis are observed

By means of an operation suggested by the author some two years ago a great number of these cases can be cured, but require the services of the experienced urologic surgeon aided by the application of the cystoscope, cysto urethroscope, and universal urethroscope to make a differential diagnosis (Fig. 215)

Prostatic Lesions.—It should not be a difficult matter to keep in mind the various lesions of the prostate, such as the inflammatory, the neoplastic and intraprostatic calculi, that may give rise to confusion when the symptoms of cystitis present themselves The rôle of the prostate is being fairly well recognized by the practitioner, and it may not be necessary to dwell for any length of time upon this phase of differential diagnosis, any further than to allude in brief to an interesting example of one form of prostatic infection giving symptoms of cystitis that are not so well known

Metastatic Infection of the Prostate, Periprostatitis, Perivesiculitis, Complicating Small Inflammatory Bone Focus

CASE XII — J. K. was said to have had an infection at the elbow, presumably an infection of an olecranon bursa, which was incised, pus having been evacuated in April, 1916 A persistent sinus followed, which did not heal in spite of a number of curettings.

August 26, 1916, the inflammatory focus was seen to extend well into the olecranon, there being bone necrosis In spite of thorough curettage the wound did not heal, but on September 29th there were symptoms suggestive of metastatic involvement of the right kidney, pain in the right hypochondrium and right region, high temperature, and pus in the urine At the

time there were symptoms of cystitis, urinary frequency, urgency and pain on urination, the prostate being tender, enormously swollen, and the region of the vesicles somewhat indurated

Retention of urine occurred, catheterization being necessary for several days, the prostate being softer in places, so that it was possible, after a week had elapsed, to express some pus through the urethra on prostatic massage

It was not until the infection of the kidney, periprostatitis, and perivesiculitis had abated that all the urinary symptoms disappeared

Urethral Lesions—The rôle of gonorrhreal urethritis in the production of the symptoms of cystitis is so well known that no further account need be given here, an interesting case of infection of the kidney and ureter having been previously described. Strictures of the urethra of large caliber may be overlooked, when these are, in truth, responsible for the symptoms of or the development of an actual cystitis

Calculus in the Posterior Urethra when Lodged Behind the Verumontanum, Having Been Passed from the Ureter, May Reside There for Weeks or Months Without Being Recognized

CASE XIII—E P, male, consulted me October 15, 1916, with the *diagnosis of cystitis*. He had had urinary frequency and the usual symptoms of cystitis, also difficulty on voiding for about one year

Examination of the bladder with the cystoscope at once suggested the previous passage of a ureteral calculus. The evidences of enlargement of the left ureteral orifice, with slight suggestion of edema about this orifice, were sufficiently characteristic to warrant the careful search for a calculus, and since this was not in the bladder, the posterior urethra was carefully inspected with a cystourethroscope, and a ureteral calculus found lodged in the fossula, just behind the verumontanum. It was an easy matter, therefore, to dislodge this, push it back into the bladder, and remove it with the author's punch forceps through the operating cystoscope.

This patient, therefore, who had been suffering with a so-called cystitis for about a year, was cured at one sitting by the application of the proper methods of diagnosis and treatment.

Extravesical Lesions.—Finally, let us pause for a moment to consider just a few of the very many extravesical causes for the symptoms of cystitis, for most of these are within the power of the practitioner to recognize, without the aid of a specialist. A routine vaginal or rectal examination, so often neglected by the busy internist, cannot fail to reveal the existence of a pelvic exudate, enlargement of the uterus, fibromyomata, ovarian tumors, carcinoma of the rectum, conditions so often responsible for the symptoms of cystitis, often not sufficiently characteristic in their manifestations to be recognized without careful palpation.

Even strangulated hemorrhoids or thrombosed hemorrhoids may give us the picture of cystitis, the latter failing to respond until a radical cure by operation is carried out.

Bladder symptoms may be the first clinical signs of the existence of a large fibroid of the uterus, or even of the existence of a pelvic exudate of tubal origin, that has been running a subacute course.

CONCLUSIONS

Let us sum up by emphasizing once more that numerous other pathologic conditions must always be borne in mind before the diagnosis of cystitis is made, that the catheter be applied early in the case of the female when the symptoms are those of so-called cystitis, that the future therapy, saving that which must be applied during the first days of acute symptoms, must not be decided upon until an exact anatomic diagnosis will have been made possible, and that the application of the cystoscope is certainly not dangerous in the chronic conditions, and should even be applied in the acute cases after the most severe stage has been passed. *With the proper agnostic attitude toward the nature of "cystitis" on the part of the practitioner, and a more intense desire to attain an exact anatomic diagnosis, much suffering will be prevented and the lives of some patients may be saved.*

all the usual symptoms of diabetes for a period of two months, and had lost 15 pounds. She had gone to a physician two weeks after the onset of her symptoms, and he, without testing her urine, told her she was suffering from "weakness of the bladder," and gave her some "brown medicine." The symptoms increased in severity, she became much weaker, continued to lose weight, and was eating large quantities of any food she pleased.

The first day in the hospital, on a general and unrestricted diet, she showed 86 grams of sugar in the twenty-four-hour period. There was a ++++ diacetic reaction, the Blood Sugar was 0.22 per cent., and the Blood CO₂ was 43 volume per cent. The subsequent course can best be followed by means of the chart.

Date.	Twenty four hour urine.		Food. Grams.				Weight.	Blood Sugar Per cent.	Blood CO ₂ Vol. Per cent.
	Sugar Grams.	Diacetic	COH.	Protein	Fat.	Calories			
Jan 3	86	++++	200+	90+	150+	2584+	125	0.22	43
" 4	24	++++	50	50	50	1075			
" 5	18	++++	15	25	25	396	124		
" 6	12	++++	0	0	0	0			
" 7	5	++	0	0	0	0	125		
" 8	0	+++	0	0	0	0		0.12	58
" 9	0	++	10	20	20	309	127		
" 10	0	++	15	30	30	464			
" 11	0	+++	15	30	30	464	127		
" 12	0	++	20	40	40	618			
" 13	0	++	25	50	50	773	125		
" 14	0	+	35	60	60	948			
" 15	0	0	35	70	70	1082	124	0.19	65
" 16	+	0	40	80	80	1236			
" 17	0	0	0	0	0	0			
" 18	0	++	20	60	60	888			
" 19	0	+	25	80	80	1075			
" 20	0	0	25	90	90	1309			
" 21	0	0	25	90	90	1309			
" 22	+	0	25	90	100	1402			
" 23	0	0	0	0	0	0			
" 24	0	0	25	90	90	1309			
" 25	0	0	25	90	90	1309			
" 26	0	0	25	90	90	1309		0.11	63
Discharged five days later on a diet of									
	0	0	40	90	90	1370	119		

You will notice on the chart that the diet of the second day was 50 COH, 50 Prot. 50 Fat, and on the following day it was reduced to 15 COH, 25 Prot. 25 Fat, while on the fourth day a complete fast was instituted, which after three days rendered the patient sugar free. The first food after the fast was 10-20-20. The exact amounts were more or less arbitrarily chosen, and 5-10-10 or 5-20-20, or 15-30-30 might have been equally suitable. Now the next question is, How shall we increase this diet in order to determine what food the patient can take eventually and remain sugar free? Experience has taught us that a gradual daily increase from the beginning of all three foods in small amounts is the most satisfactory method in the average case. Some patients, of course, immediately after the fasting period are not able to tolerate as much as 10 grams of COH when given without any other food, while others tolerate as much as 30 COH, 60 Prot. 60 Fat the day after the fast. These figures, of course, represent extremes, but in the early days of treatment, that is, when the patient has been made sugar free for the first time, there is no definite way to determine in advance just what the immediate tolerance for food will be. The Blood Sugar is usually the best guide, but this is not always reliable unless we do repeated Blood Sugar determinations on the first feeding day, a procedure which is not always practicable, nor is it necessary.

You will notice in this case that the Blood Sugar was 12 per cent. on the first day following the fast. This suggested that the diet could be increased rather rapidly and, as shown in the chart, this assumption was correct, for after eight days in which the diet was increased daily, a tolerance of 40-80-80 was reached when sugar reappeared in the urine. The Blood Sugar at that time being 0.19 per cent. A second fast day was then given, and a diet of 20-60-60 followed. This lower diet, in turn, was increased to 25-90-100 when sugar reappeared, and then following the next fast day 25-90-90 was tried. On this the patient was sugar free for three days. Then following a gradual increase of COH (5 grams per day), keeping the Prot. and Fat

at a constant level, a food tolerance of 40—90—90 was attained, on this diet the patient was discharged

She returns to the hospital today having kept this diet for the past two weeks and having taken one-half diet day once a week according to directions. She has not shown sugar or diacetic in the urine since her discharge and her weight is stationary. We will now instruct her as to her future treatment.

What guide have we in telling her whether or not

- 1 To increase the diet?
- 2 To keep the same diet? or
- 3 To decrease the diet?

MR P The Blood Sugar will help, as will also her weight.

DR. GEYELIN Yes, and the Blood Sugar has been done. It is 0.125 per cent (four and a half hours after her last meal), which was breakfast. With such a Blood Sugar, and the weight far below normal, it is probably safe to give a slight increase in the food. How much would you give? and what food or foods would you increase?

MR R I should think she needed more calories, and a slight increase in the fat would be the best way to give it—say 10 grams

DR. GEYELIN That would probably be the most advisable. But why not increase the protein or carbohydrate?

MR. R The patient is now receiving more than 1½ grams of protein per kilo body weight, and, as I understand it, this is the most generally accepted quota of protein for adults on a mixed diet. As for the carbohydrate, it seems to me that the patient is receiving enough for the time being in view of her original low carbohydrate tolerance and the present Blood Sugar level, which is at the upper normal limit (fasting)

DR. GEYELIN Yes, I think that is good reasoning, because an increase of 10 grams COH would be more apt to cause glycosuria during the next two weeks than would an increase of 10 grams of Fat during the same period, and yet might it not be possible to give 5 or 10 grams more of COH?

MR R Yes, sir

DR. GEYELIN Some might think it best to give even more of

an increase than we have suggested, but experience teaches us that gradual small increases in the food with the consequent probability of keeping the patient continuously sugar free, particularly during the early months of treatment, is the best procedure. By this method it has seemed to me that the patient is more likely to feel better, more likely to gain a better tolerance eventually, and, further, it is stimulating to the patient's morale to see a steadily increasing diet without the reappearance of sugar. Of course, you must all remember that if the body weight is falling off rapidly and the patient is getting weak, in spite of restricted activities, the diet must be increased more rapidly, as this additional loss of weight in itself may be the indication of a diet too far below the patient's tolerance.

The question of whether or not to leave this patient's food as it is, or to decrease the present amount, it seems to me is answered by the foregoing discussion. In other words, when a patient has been taking a given amount of food without showing sugar for two weeks or more, and the Blood Sugar continues to be normal or nearly so, and there is no gain in weight, there is certainly no danger in increasing the food as long as the patient is under proper supervision. This supervision should continue at two-week intervals for many months. In the more severe cases patients should return at least once a week to have their food intakes regulated. Before dismissing this patient there are several other details of the treatment that demand attention.

Some of you may have wondered why, on the first day of the patient's stay in the hospital, we allowed her to continue on a full diet. This, of course, would not be permissible if there had been any dangerous symptoms, such as those of marked acidosis or of oncoming coma, but there were none, and, as I will show you, we gained much useful information by observing how much sugar was excreted in twenty-four hours when the patient was eating the amount of food shown by the first figures on this chart.

Let us assume, for instance, a much more severe case of diabetes, one who on a diet of about 150 grams COH is excreting sugar in amounts exceeding 200 grams daily, this would indicate a negative COH balance, and the probability that sugar was

being formed from some other source than the COH of the food. That is, either from stored COH, which is not likely, or from food and tissue protein. This negative COH balance in itself is presumptive evidence of a more severe form of diabetes than the case I have just shown you.

You have probably also wondered why we did not put the patient on a fast period immediately. The reason for not doing so was that occasionally, particularly in diabetes complicated with nephritis or obesity, if a fast is initiated at once there is danger of causing acidosis, or increasing an existing one, so that a gradual reduction of the food to the point of actual fasting is the safer routine measure. Dr. Joslin, who has had great experience in the treatment of diabetes, believes in the following routine method of reducing the original high diet to the fasting level. On the first day "omit fat, after two days omit protein, and then have the carbohydrates daily until the patient is taking only 10 grams, then fast." This method of reducing the diet requires more time, and has no marked advantage in other respects over the method shown on this chart, furthermore, I have never seen any unfavorable results when the food was reduced in the manner I have shown you in the chart. The important underlying principle being not to plunge the patient from a high fat diet into an immediate fast day.

During the first part of the clinic today I told you that one of the most valuable contributions to the treatment of diabetes that Dr. Allen has made was his study of the ill-effects produced by long-continued high fat feeding, particularly when the carbohydrates were reduced to what was formerly termed the "carbohydrate-free diet." This so-called carbohydrate-free diet usually consisted of 10 to 20 grams of carbohydrate, 80 to 150 grams of protein, and from 150 to 250 grams of fat. It is quite true that on such a diet patients often remained free from sugar for periods ranging from many weeks to many months, but without any further change in the diet they then began to show sugar in increasing amounts. This was usually foreshadowed clinically by general languor, weakness, distaste for food, sometimes with actual nausea and diarrhea. There were pains in various parts

of the body, particularly in the extremities, and sometimes periods of unusual drowsiness

If at this stage the patient consulted his physician, it was usually found that small amounts of sugar were present in the urine or there might be no glycosuria. In either case, however, traces or considerable amounts of diacetic acid were to be found in the urine. Not realizing the real significance of what was happening, namely, that the patient was getting too much fat, the physician, as a rule, increased the carbohydrate because of its antiketonuric effect, but maintained the fat intake at its previous high level. This procedure, of course, immediately caused an increase of the Glycosuria and temporarily decreased the amount of Diacetic Acid excreted. Intermittent periods of "green days" and in some instances "oatmeal days" were resorted to for the purpose of decreasing Glycosuria, but a return to the high Fat diet invariably followed, and this, in turn, increased the ketonuria. The underlying principle of treatment that prevailed at that time was that the patient's nutrition must be maintained at any cost and that he must not be allowed to lose weight. How false this theory was can be seen from the very unfortunate results that followed such treatment, particularly when contrasted with the very favorable results obtained with our present methods.

Nowadays, in such a case, armed with the experimental proof of the harmful effect of high fat diet on the diabetic, we take the Blood Sugar which, under these circumstances, is invariably high, and with it there is almost always a lowering of the Blood CO₂. Immediate reduction of the fat, followed by a period of fasting, or at least a period of low diet, especially one low in fat, will in time lower the Blood Sugar, raise the Blood CO₂, and render the patient free from sugar and diacetic acid. Frequently this takes many weeks to accomplish, and during that time it will be found that the tolerance for all foods is much lowered.

I will now show you a case which I think aptly illustrates the beneficial effects of modern treatment. This patient was suffering from a prolonged period of a diet too high in Fat

The patient, a man aged forty-eight, first came for treatment in October of the past year (1917). He had had diabetes for two years previously. Up to June, 1917, he had treated himself. He then went under the care of a physician, who found that he had a good deal of diacetic acid and sugar, together with a high Blood Sugar. He was immediately put on a fast, which rendered him sugar free, the diet was then slowly increased until he was eating approximately 60 grams of carbohydrate, 100 grams of protein, and 180 or more grams of fat, and was still free from sugar. For one month on this diet he did well. He was free from sugar and gained slightly in weight and strength. The Blood Sugar remained normal. Shortly after this, in August, he began to feel "good for nothing." He would often sleep the greater part of an afternoon, felt weak, began to lose a little weight, and also had considerable pain in his legs and feet, particularly at night. Finally, in September, he began to have traces of sugar in the urine. He at first took fast days to clear this up, and then, as he was feeling worse, he began to eliminate glycosuria, when it occurred, by reducing the carbohydrate alone, leaving the fat and protein at their usual level. He did this repeatedly for two or three weeks, and then began to feel badly, was weak, had much pain in his feet, and for the first time he began to notice that his eyesight was rapidly failing. It was at this time (October, 1917) that he first came to the hospital for treatment.

As soon as he came under treatment it was found that his Blood Sugar was over 0.2 per cent, that his Blood CO₂ was 40 volumes per cent. He was excreting considerable traces of sugar in the urine, and had a ++++ diacetic reaction. The accompanying chart shows the progress of the case as far as the intake of carbohydrate, protein, and fats are concerned, together with the Blood CO₂, the Blood Sugar, and the weight.

It took more than three months before any change was noticed in his clinical condition, in fact, at first he grew weaker and his eyesight became more dim. His tolerance was increasing very slowly from a very low level, but was not nearly as high as it had been during the preceding summer. The only improve-

ment was that he no longer had pains in his legs and feet. In spite of the reduction of the fat, he apparently could tolerate no more carbohydrate than when he was getting a high fat diet. For the past four weeks, however, he has been steadily improving, he is much stronger, takes more interest in life, and his eyesight is much better, in fact, he says that it is almost as good as before the dimness of vision began. He can now tolerate 65 grams of carbohydrate, and this with a low Blood Sugar.

The immediate treatment instituted was as follows, showing the urine and food of the last day (October 16) before treatment was begun.

Date	Twenty-four hour urine		Food Grams.				Weight	Blood Sugar Per cent	Blood CO ₂ Vol. Per cent
	Sugar Grams	Diacetic	COH.	Protein	Fat	Calories			
Oct. 16	22	++++	25?	100+	200+	2300+	141	0.28	40
" 17	20	++++	25	50	50	750			
" 18	17	++++	10	30	30	430			
" 19	5	++++	10	20	10	210	140		
" 20	++	++++	0	0	0	0			
" 21	+	++++	0	0	0	0			
" 22	0	++++	0	0	0	0	143	0.17	52
" 23	0	+++	5	10	10	150			
" 24	0	+++	10	20	20	300			
" 25	0	+++	15	30	30	450	147		
" 26	0	+++	20	40	30	510			
" 27	0	++	20	40	30	510			
" 28	0	+++	25	40	40	530			
" 29	+	+++	30	40	30	550			
" 30	0	+	0	0	0	0	140	0.15	58
" 31	0	++	20	40	20	420			
Nov. 1	0	++	25	40	20	440			
" 2	0	++	30	40	20	460	137		
" 3	0	+	35	40	20	480			
" 4	+	+	40	40	20	500			
" 5	0	+	0	0	0	0			
" 6	0	+	30	50	30	590	135	0.13	62
" 26	0	0	30	90	70	1110	138	1.3	65
Dec. 26	0	0	50	90	70	1190	138	1.4	60
Jan. 25	0	0	55	100	80	1340	137	1.3	60
Feb. 26	0	0	65	100	80	1380	136	1.2	60

I must supplement the figures in the chart by saying that once every two weeks during December, January, and February the patient received a routine fast day, whether there was glycosuria or not, also, that a half diet day was given when glycosuria appeared in the interval between fast days. In this case a half diet was always sufficient to clear up glycosuria.

What the eventual tolerance will be is hard to say, but judging from experience with similar cases it will probably be possible to raise the food to 80—100—120, and still keep the patient free from glycosuria. This may take several months, but if every precaution is taken we will probably reach this tolerance. A rapid gain in weight of 5 or 10 pounds, a steadily rising Blood Sugar, or an increasing acidosis will be the signal that we are increasing the diet too rapidly. In that case, and if it is impossible to get a higher tolerance than the present one, the patient must regulate his general activities to his caloric intake.

In other words, we must not force the calories upward simply in order to make it possible for the patient to lead a normal life. It is far better to let such patients understand that they are not normal, and that for the sake of possible eventual improvement they must live at a level of metabolism where the caloric output is commensurate with the caloric intake. A few cases do eventually show considerable improvement under this method of treatment, and certainly it is the only known method that offers any hope of ultimate success.

If we ever expect to improve, and perhaps in some cases cure, the pathologic condition that causes diabetes we must, first of all, keep our patients free from the first and cardinal symptom of the disease, namely, glycosuria.

It must be borne in mind that there are a few cases where freedom from sugar is not practicable nor desirable. Such cases are, fortunately, rare, and they are the very severe diabetics where the diet necessary to keep the patient sugar free is so low that even with complete rest in bed the calories supplied by the food are not sufficient to prevent increasing weakness and rapid starvation. When this condition confronts one it is better to give sufficient food to maintain a moderate degree of

strength and allow the presence of sugar in the urine. The diet in such cases must be carefully watched and regulated so as to guard against severe acidosis and too great an increase of sugar in the urine.

Patients in whom such severe diabetes occurs are usually under thirty years of age, although older diabetics are not exempt.

CLINIC OF DR JESSE G M BULLOWA

WILLARD PARKER HOSPITAL

LOCAL EVIDENCE OF TONSIL INVOLVEMENT IN THE CAUSATION OF DISTANT OR SYSTEMIC DISEASE

Significance of Focal Infection to Distant Disease Case Reports Deforming Arthritis with Renal Insufficiency, Mitral Stenosis and Infarct of Lungs Chorea with Low-grade Fever, and Salpingitis. Criteria of Adequate Treatment. Enlarged Lymph-nodes and Other Stigmata of Absorption. Advantages of Local Anesthesia in Tonsil Operations.

So much has been written and so much evidence has been successfully adduced to show that the tonsils may serve as a portal of entry for general infections that the mere narration of additional cases will serve no purpose. The term "focal infection" has begun to take the place of the term "cryptic infection," and the word "cryptic" has reverted to its ancient meaning. Under this standard have been gathered many victories against chronic disease. This slogan, however, has not been popular in all quarters, and so long as it remains a catch phrase that is not properly related to that ancient medical doctrine, *ubi pus, ibi evacuo*, it is bound to work havoc in the hands of routineers.

Much opposition has been aroused against the entire doctrine of focal infections as a result of unnecessary treatment or by imperfect treatment of a focus. Throats have been incompetently operated upon for tonsil infections, but the infection has not been eradicated, and then the procedure instead of its inefficient execution has been blamed.

The type of cases which illustrate what is meant by distant disease are the following:

1. Margaret F came under observation at the age of twenty-three, having been bedridden with deforming arthritis for two years. Her jaws were partially ankylosed and she was extremely wasted. Her eyes were prominent and the backgrounds showed hemorrhages. The thyroid was slightly enlarged and there was universal enlargement of all lymph-nodes. She had a pre-systolic murmur at the apex of the heart. Her systolic blood-pressure was 200, there was urinary evidence of nephritis, and she had had hemoptysis. She was carefully studied at the City Hospital, where she died a short time later of a true renal insufficiency with a blood non-protein nitrogen of 240 mg. The autopsy revealed a tight mitral stenosis, evidence of former infarcts in the lungs, and kidneys shrunken to the last degree as the result of successive inflammatory invasions. The essential fact in the history was that at the ages of sixteen, seventeen, and eighteen she had been an inmate of one of the best hospitals in the city suffering from chorea, which was treated by the accepted methods. She had had frequent attacks of sore throat, but her tonsils had never been operated upon, and at the time of her later illness they were small and buried. In seven years this girl had been destroyed by successive invasions of her tissues from her focus, so that at the age of twenty-three she presented the appearance of advanced age clinically and pathologically.

Contrast this case with these others

Alma H had her tonsils operated upon inefficiently several times in childhood. At thirteen years of age she developed an acute chorea associated with an acute infection of the tonsil stump. After several weeks of unavailing treatment with salicylates and arsenic, the tonsils were enucleated by Dr. Henry L. Lynah. The chorea and the low-grade fever terminated within a few days, and there has been no recurrence of either in four years, in spite of all the conditions in the home and diet remaining unchanged.

Leonora A, sixteen years of age, at high school, was suddenly taken with a disabling attack of pain in the right lower abdominal quadrant and local symptoms of visceral inflammation, *i.e.*, muscle spasm and hyperalgesia, associated with a rise in tem-

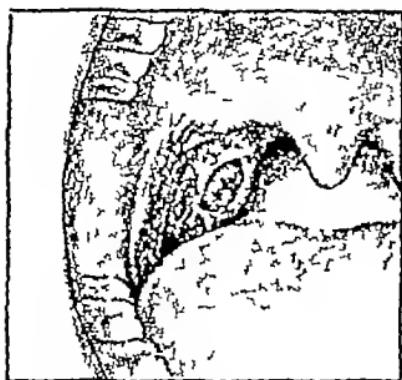
perature to 102° F. Rectal palpation revealed an induration to the right of the uterus. Dr. Charles G. Childs, Jr., confirmed the diagnosis of salpingitis. From the very small tonsil stumps left after tonsillotomy under general anesthesia seven years previously pus could be expressed. The regional lymph nodes were large, and each expression of pus from the tonsil was followed by slight aggravation of the symptoms for a few days, but with subsequent marked improvement lasting several weeks. Tonsillectomy under general anesthesia by Dr. Lynah was followed by complete and permanent cure of the condition, disappearance of the nodes, and cessation of the attacks of inflammation in the tube. When enucleated the tonsils were found to be much larger than had been anticipated, and they contained numerous discrete pus pockets.

These conditions may be explained either as due to the absorption of bacteria, which form infarcts, or by the absorption of the products of bacterial life or death, which act selectively and change the dispersity of the proteins in certain sensitized regions. There is another explanation possible, and that is, that the foci mechanically or chemically irritate the autonomic nervous system, thus producing distant changes.

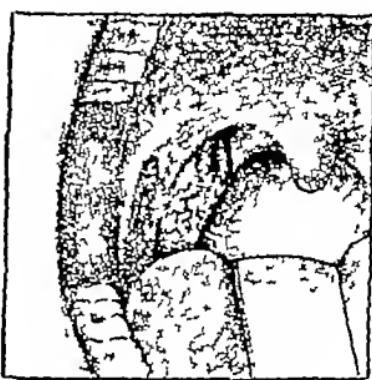
Bacterial evidence has been relied upon by some as the criterion which shall determine surgical treatment of the tonsils. Clinicians of eminence have determined upon tonsillectomy, basing their judgment solely upon the result of cultures. To my mind, this is an unsafe guide, because we have learned at the Willard Parker Hospital to value more highly, for instance in the case of diphtheria, the clinical picture which is the body's response to the irritant than the mere finding of the Klebs-Löffler bacillus. Annually we see a number of cases in which antitoxin has been withheld too long from diphtheria patients because no diphtheria bacilli have been reported from the laboratory. And this in spite of the fact that the body itself, by its reaction, local and constitutional, should have demonstrated to instructed eyes the nature of the disease. Nor is the positive finding of diphtheria bacilli, even though virulent, a guide in the administration of antitoxin. The persistent carrier

state and the Schick reaction have demonstrated that diphtheria bacilli may exist in the absence of the disease diphtheria

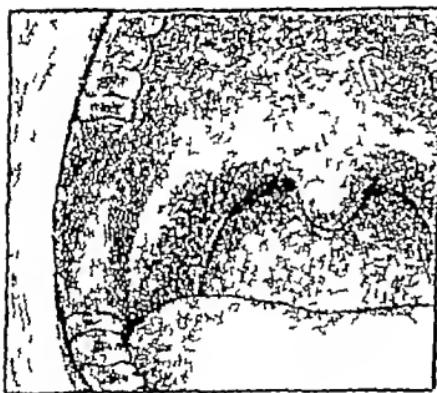
If this analogy is valid, it is worth while to inquire what is the local evidence of tonsil infection which may be associated



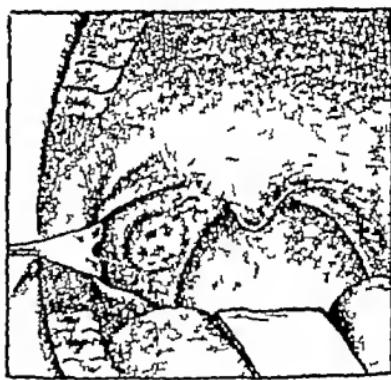
1



2



3



4

Fig 216—Types of diseased tonsil 1, Diaphragm appearance of pillars which interferes with crypt drainage, 2, maldevelopment of lower tonsil segment, inflamed plica, with suprapical tonsil folded on itself and surfaces fused, 3, inflamed and prolonged mucous membrane fold which conceals tonsil even when patient gags, 4, small buried tonsil, which is only revealed when anterior pillars are retracted

with distant or systemic disease. We realize that it might be, and to a certain extent is, possible to differentiate clinical pictures in accordance with the particular invading organism, but we have attempted no such refinement here. This problem has engrossed me for a long time, and it seems to be one that has

not received the attention it deserves at the hands of the medical profession. There is no special examination required of medical men which is more often performed perfunctorily than the examination of the throat. There are men who carry stethoscope and ophthalmoscope who draw inferences concerning the health of tonsils that they have never seen

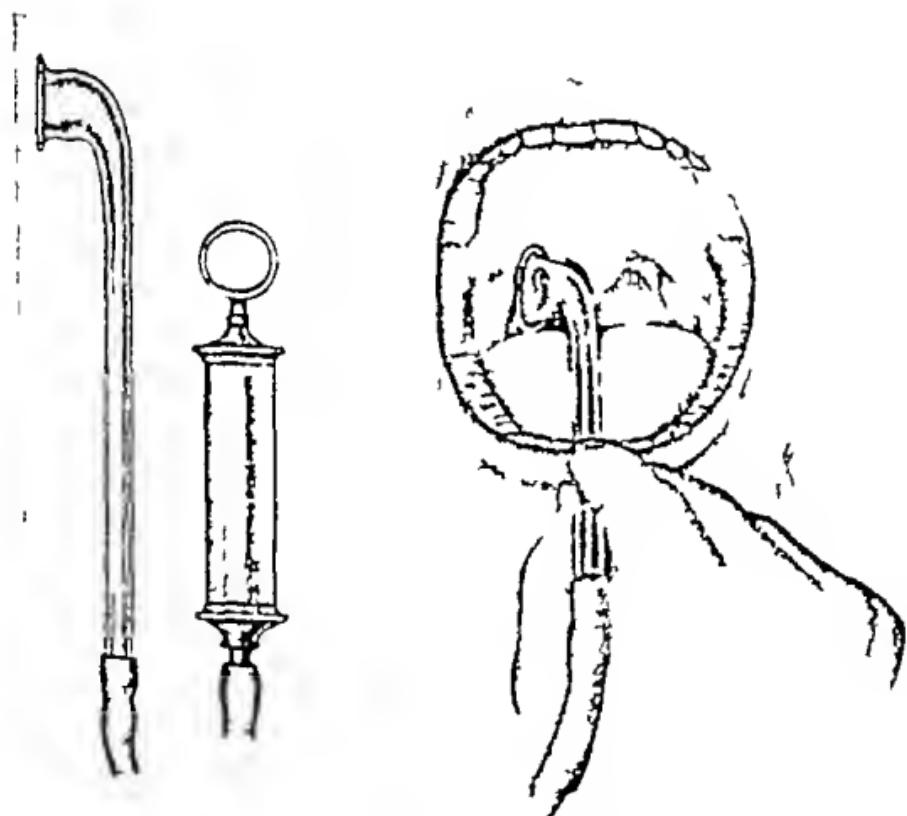


Fig. 217.—Method of determining condition of tonsillar crypts by suction with simple apparatus. Pus exuding from crypt.

The problem is one which more often confronts the internist and general practitioner than the nose and throat specialist. Patients who have sufficient local symptoms to seek the aid of the specialist independently have subjective evidence of the source of their trouble. They come, as it were, labeled

These strictures on the usual examinations are made in spite of the improvement resulting from the popularization of the illumination of the oral cavity since the introduction of cheap and

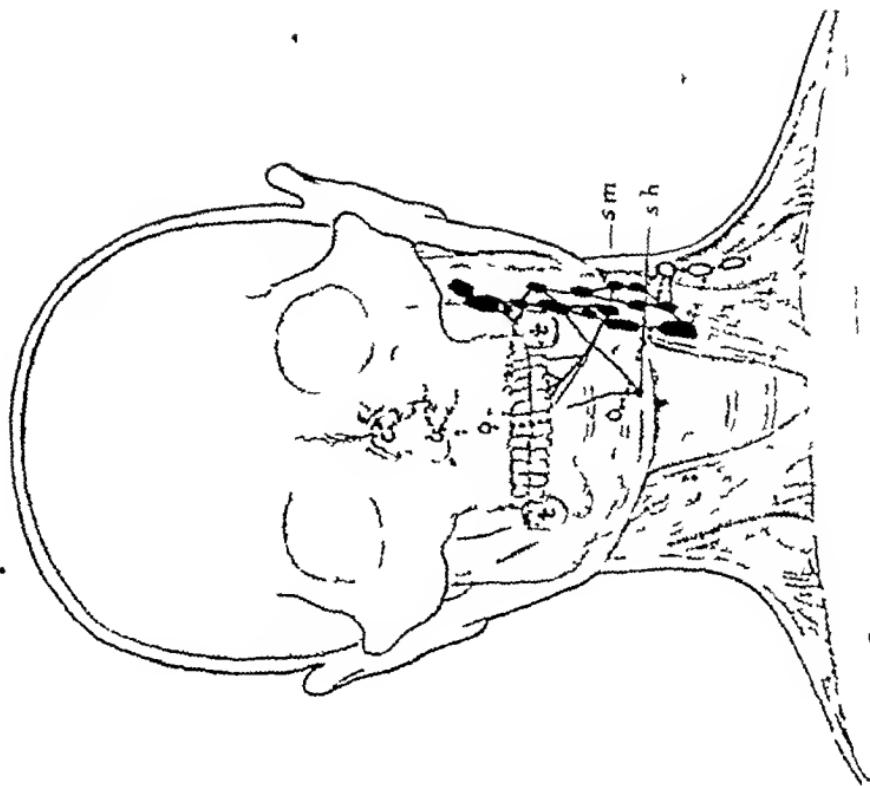
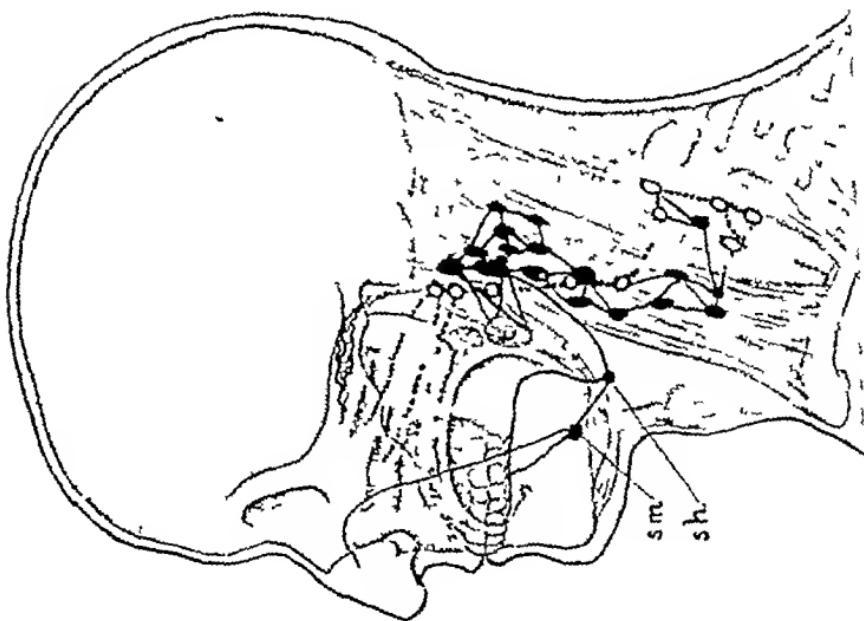


Fig 219



portable flash-lights. Frequently patients will report that they have been told that they have no tonsils, or healthy ones, when on questioning it is evident that it must have been impossible for the examiner to have seen the tonsils in his examination. The difficulty in viewing them may have been due to a high standing tongue which was not depressed, or it may have been due in patients able to depress their tongues, and these have frequently been great garglers, to the fact that there had been a prolongation of the mucous membrane covering the anterior pillar so that it completely covers the tonsils. Such tonsils may be bent upon themselves so that the two surfaces are in contact. These surfaces may fuse if, as the result of irritating discharge, granulations replace epithelium (Fig. 216, 2)

Another type of buried tonsil is accounted for by the fact that the tonsil develops by the fusion of two portions, thus forming the plica or fold, where the lower segment is poorly developed or absent, the smooth plica may bury the upper developed portion. It is quite obvious that a view of the smooth plica may give little evidence of the condition behind it. To examine the tonsils it is necessary to see them and determine whether they are healthy and whether their crypts are emptied of detritus by the constrictor action of the anterior and posterior muscular pillars. The normal tonsil is easily emptied of detritus, and it is only when the plica fuses with the posterior pillar and forms a recess that there is retention in the upper crypts. At times the tonsil enlarges, but does not protrude between the pillars, they cover it like an iris diaphragm and retain the secretion (Fig. 216, 1)

Fig. 218, 219.—Anterior and lateral of main lymph drainage areas in the head and neck prepared from clinical studies & Tonsils 5 m., submaxillary node 2.5, suprathyroid node. Solid network above superior turbinal shows drainage with meningeal system, inoculating with retropharyngeal nodes. Dotted lines and white nodes show drainage of upper teeth deep face extend to level of hyoid and then in posterior triangle. Solid lines and black nodes show drainage of tonsils, lower teeth tongue and superficial face. The deep and superficial chain both inoculate and empty into subclavian vein. The lymph-nodes of the tonsil are indicated to be superior and behind the tonsils, diverging in this respect from the usual diagrams.

A type of tonsil which causes trouble is the so-called small cryptic or buried tonsil (Fig 216, 1) I am frequently asked how it is that many people have had no trouble with their tonsils and nevertheless develop degenerative conditions at middle age which are attributable to them There are two possible explanations either the tonsils have been diseased for a long time and the systemic condition suddenly becomes sufficiently severe to attract attention, or, with the atrophy of lymphoid tissue incident to aging, the tonsils shrink so that they are no longer sufficiently large to be compressed by the pillars In some throats the upper portion of the tonsil may be grasped and emptied, while the lower follicles may be the source of infection

To view the tonsils properly requires a depression of the tongue, and in many cases the patients must be made to gag in order to throw the tonsils into the field of vision The tonsils lie on the inner wall of the pharyngeal tube and do not naturally, in normal swallowing, face forward Frequently, in order to expose the tonsil, it is necessary to retract the anterior pillar or the plica with a hook or retractor No examination of the tonsils is complete unless the condition of the crypts has been determined This can be done by suction or expression (Fig 217), or by observing the destructive effect of irritating discharges

When the head is slightly flexed the tonsils lie opposite the ramus of the jaw, and if, after the tongue is depressed, the tongue depressor is brought up against the pillars from below with pressure against the jaw, pus may frequently be expressed from the crypts and the supraphical pocket, into which some crypts may empty There is no other way, to my mind, of determining whether the crypts on the superior aspect of the tonsil are emptied by the normal swallowing movements

The inability to express pus must not be taken as evidence of its absence from crypts whose openings may have become occluded Pus sometimes dissects its way into a pocket upon the capsule behind the tonsil The absence of detritus is not evidence that the tonsil is healthy The inflammation may be diffuse

Several times I have seen tonsils which were described as sclerotic, and from the whitish mass which prompted the inference thick pus has been evacuated. Diseased tonsils may show either an increased redness and an increase in the lymphoid tissue overlying them, or they may show fusion of the pillars (and these are particularly deceptive), or there may be fusion of the tonsil with one or the other pillar. An irritating discharge from the crypts may destroy the epithelium about their orifices. The anterior pillars are frequently covered with a fine network of small vessels. If the patient has been unfortunate enough to have smoked, the condition will surely be called "smoker's catarrh."

The evidence of pus or, in other words, the evidence of infection of the tonsil is the same as that of infection anywhere in the body. The main reliance should be placed upon the evidence furnished by lymph node involvement. The path from the tonsils to their regional lymph nodes is not as long as that from the fingers to the epitrochlear or axillary lymph nodes, nor is its path streaked with visible red. It is not as well known nor is its importance appreciated. A knowledge of the location and drainage areas of the neck is of very great importance as an aid to diagnosis, and is not sufficiently understood. (See Figs 218, 219.) So important is the condition of the position of the lymph-nodes involved in infections about the head and face that its clinical significance cannot be overemphasized. By observing the regional lymph node involvement patients will be preserved from many unnecessary x-ray exposures and directed to those which are necessary. The inference drawn from roentgenograms of the teeth should be fortified by clinical observation of the lymph-nodes involved. I shall digress for a moment to discuss the drainage areas of the face and throat.

The tonsils drain into the deep lymphatics of the neck which lie on the outer side of the jugular vein. When these are inflamed they are best palpated high up in the neck above the level of the tonsil, by compressing them against the transverse processes of the vertebrae with the thumb or index-finger (Fig. 220). They are to be felt in a triangle formed by the posterior be-

of the digastric and the sternomastoid muscles. They may be located best by palpating first the pulsations of the carotid artery. They are situated a centimeter or less to the outer side of the artery, for they rest to the outer side of or upon the jugular vein. The fingers may be hooked about the sternomastoid muscle and the nodes may be grasped between them if desired.



Fig. 220.—Method of palpating deep cervical glands with thumb against vertebral column high up in the triangle formed by posterior belly of digastric and sternomastoid muscles. These glands drain from tonsils, tongue, and lower jaw.

Frequently they are tender, and they may be followed down the course of the vein. The head should be slightly flexed, both forward and laterally, and turned to the side being examined during the maneuver (Fig. 220).

These deep cervical nodes drain the lymphatics from the face, tongue, and teeth of the lower jaw. If the face or teeth of

the lower jaw are involved, a gland situated just inside of the mandible and behind the groove of the facial artery is enlarged. If the mandibular or tongue area is involved, the so-called gland of the tonsil, or gland situated behind and below the angle of the jaw, is enlarged. In my experience it is the exception rather than the rule for this gland to be much enlarged in tonsillitis.

When the teeth of the upper jaw or the sinuses are infected the upper portion of the deep cervical chain usually escapes. These areas drain into retropharyngeal glands situated behind the pharynx, and thence into the lower portion of the deep cervical chain which lies under the sternomastoid muscle below the level of the larynx. This chain becomes superficial behind the sternomastoid, and sometimes, in order to protect it from pressure, the shoulder is raised, thus relaxing the muscle. These patients frequently complain of pain in the arm at the insertion of the deltoid as a result of the strain upon that muscle. The removal of pus from a point in the area whence they drain is frequently followed by very speedy relief of pain in the muscles of the arm. At times the retropharyngeal glands may be palpated when the larynx is moved aside and they may be visible through the pharyngeal wall, sometimes they are covered with adenoid tissue. The area above the uppermost turbinal bone drains with the meningeal system.

In discussing absorptions from the tonsils we have so far considered only such stigmata of absorption as may occur from the existence of pus at any point in the body, namely, lymphatic node enlargement, redness, swelling, tenderness, and pain. These patients may have frequent recurrent attacks of discomfort in the throat which they do not interpret as pain because the surrounding tissues are so unusually lax for the purpose of permitting free movement. But there are other local symptoms which are due to the fact that the tonsil is an organ, these are the referred zones of hyperalgesia.

For a long time I have been observing the areas of herpes associated with acute and chronic tonsillitis, and have come to believe that the popularly called "fever blisters," if they are situated at the vermillion border or slightly above or below it and near the

angle of the mouth, especially if external to the line of the philtrum which indicates the point of fusion of the nasofrontal and maxillary processes, are due to distensile pressure in some crypt in the tonsils. These herpes are preceded and accompanied by a hyperalgesic zone to pin scratch which is quite definite. If the skin is blanched by stroking with a pin or with the finger,

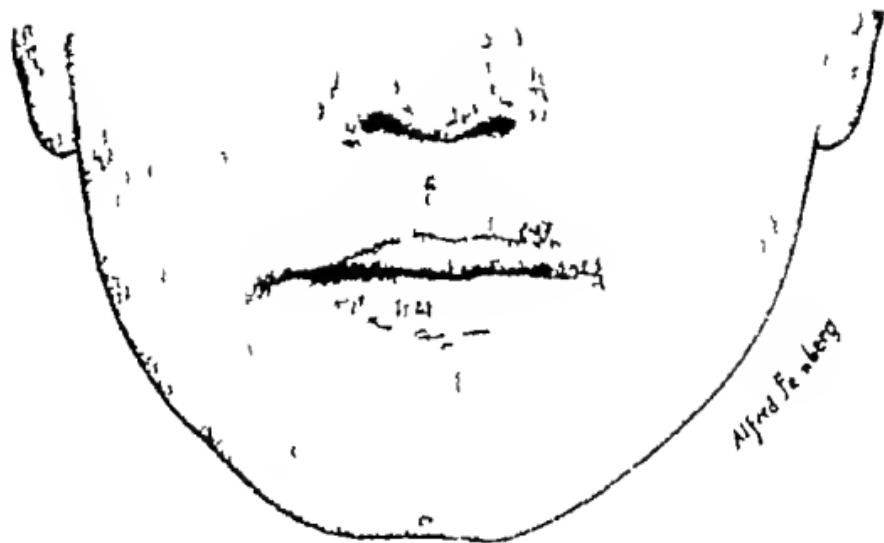


Fig. 221.—Location of herpes associated with distention of most superior crypts of tonsils (described in text)

the herpetic zone may be prefigured by dilated capillaries over the area corresponding to or prefiguring herpes

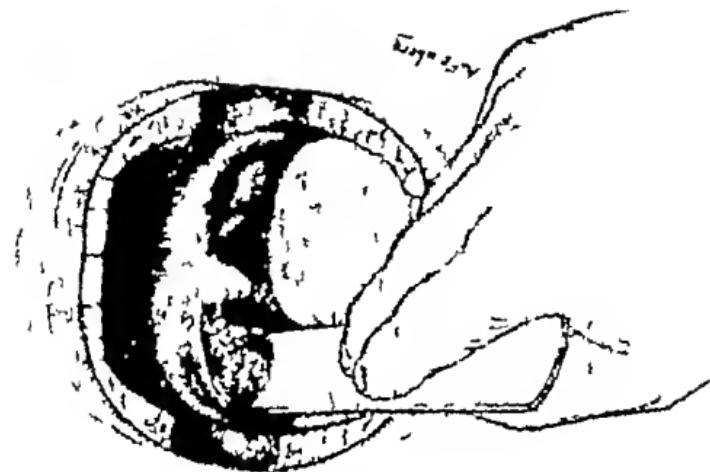
These areas are the anterior portion of the tonsil zone, and I have learned to look at the upper portion of the tonsil for distended crypts when they occur upon the upper lip, and at the lower portion when they occur upon the lower lip. The posterior

PLATE 1



Herpes on lip associated with distention of upper tonsil crypts shown in Plate 2. On the right side of face capillary dilatation in blanched area induced by stroking lip with pin corresponding to area of hyperalgesia and often prefiguring herpes.

PLATE 2



Exposure of diseased tonsil by gagging and method for expressing pus from crypt emptying into the suprtonsillar fossa.



Inflamed appearance of tonsil which frequently reverses a diseased tonsil

tonsillar area is the one usually figured, it is a small triangular zone which lies just posterior to and above the thyroid cartilage. Several times I have found a small area of herpes or of hyperalgesia on the cheek just above the nasolabial fold. This has been associated with a distention in the most superior crypt under the phloca (Fig. 221).

The correctness of these inferences is also attested by the observation of cases in which herpes ceased to occur after tonsil enucleation, and by several cases in which the occurrence of lateral labial herpes was terminated by tonsil resection, but the philtral herpes continued to appear with subsequent maxillary inflammations.

The hyperalgesia sometimes comes into consciousness and is interpreted as neuralgia of the throat or face, but this must be distinguished from the recurrent attacks of pain in the ear which may be due to closure of the eustachian tube from edema, for these latter have the same basis as the recurrent colds in the head which occur in tonsillitis as the result of lymph stagnation, or stoppage brought about by the swelling of the lymph nodes. I have had occasion to observe marked induration of lymph nodes in a furuncle of the neck associated with congestion and increased discharge from the same side of the nose.

Let us enumerate the local symptoms of tonsil infection. Recurrent attacks of pain from distention or infiltration, redness and swelling of the tonsil and peritonsillar tissue, and I should add to these the evil or putrid odor which may be due to the growth of *Streptococcus putridus* or to decomposition of detritus, destruction of the tonsillar epithelium by an irritating discharge and its replacement by granulations, regional lymph node swelling and tenderness which may occasionally cause blocking of other areas, as, for instance, the nose or eustachian tubes or larynx when the involvement extends down sufficiently to take in their drainage areas, and referred sensitiveness to pin scratch or hyperalgesia with a capillary dilatation which may be so severe as to manifest itself as herpes.

It is pertinent to remark that competent treatment of the tonsils requires that these local signs of tonsil inflammation shall

entirely disappear as a result of operation on the tonsil, and it has been my experience that where they have not been relieved the tonsil operation has been inefficiently performed and a small crypt-containing mass has remained in the upper pole or near the base of the tongue. This is especially disastrous if the tonsillar pillars have been mutilated during the operation. For example, a little girl had her tonsils removed by a competent operator who left a small segment in an upper pole. The indication for the original operation was frequent recurring colds in the head. The posterior pillar was cut away. Three years later the remnant of tonsil became infected and was the cause of a severe, acute polyarthritis which terminated abruptly when the remnant of tonsil was removed and about 1 c.c. of pus evacuated. This procedure was undertaken because of the persistent large and tender regional lymph-nodes and the redness and infiltration of the anterior pillar.

The results of the removal of infected tonsils are so brilliant that frequently the procedure should be offered to patients who are otherwise unfavorable anesthetic risks, for there are a number of advantages in local anesthesia for tonsil operations. The irritant action of the anesthetic on the lung tissue is avoided, there is no danger of the inspiration of infected material or blood with its attendant risk of lung suppuration or pneumonia, the danger of hemorrhage in an unconscious patient is avoided, as the patient can co-operate and give the alarm. In some diseases it is a serious matter to even temporarily change the physical state of the body lipoids. For the operator, the position of the patient is that in which the surgeon is accustomed to examine and the landmarks are familiar. The performance of the operation under local anesthesia requires a simpler technic, though a greater degree of dexterity, than under general anesthesia.

INFLUENZA OF THE HEAD AND CHEST

Outstanding Clinical Features Stimulation from the Viewpoint of Colloid Chemistry Individual Symptoms and Their Specific Management. Value of Special Drugs Causes of Death and Valuable Prognostic Signs Fallacy of Drastic Catharsis and of Forcing Fluids

I SHALL not attempt at this time to discuss the prevention of infection by influenza, for testimony is very conflicting as to the value of various procedures. At this hospital we have relied upon the use of gowns and caps and careful washing of the hands after contact with the patients. Masks and cubicles were employed in a single ward only, but comparable data is difficult to obtain. All people are not susceptible to influenza and, as William H. Park has shown, any measures which are adopted after the peak of the epidemic has been passed will then seem to prove favorable.

As we observe the patients, the outstanding feature is their cyanosis or dusky complexion. Nearly every individual who suffers from influenza has a florid complexion. This was so pronounced in the first sailors that were brought ashore into this hospital in September, 1918, that they looked like mulattoes. It was also particularly noticeable in a company of marines who had enlisted from the northwest and who were the descendants of Scandinavians, their swarthy complexion contrasted sharply with their light hair and blue eyes.

It is not only the color which is characteristic of influenza patients, but the puffiness of the tissues, they seem congested. This reaction of the tissues seems to be due to the Pfeiffer bacillus. Some experiments which I performed at this hospital with the assistance of Dr. Skeers are interesting. We injected intracutaneously 1 or 2 minims of a vaccine of Pfeiffer's bacillus in several groups of cases. In susceptible patients, especially those

in the early stages of influenza, there was a very intense local reaction. The skin about the site of injection became swollen and violet colored within twenty-four to thirty-six hours, and frequently there was an accompanying enlargement of the regional lymph-nodes. Control injections of streptococcus vaccine in these patients gave at most a small pinkish area of infiltration. The influenza vaccine reaction was hemorrhagic and it persisted for a number of days and then subsided, leaving the skin stained. Though this does not by any means prove the etiologic relation of the Pfeiffer's bacillus to the clinical entity known as epidemic influenza, nevertheless the perfusion of the tissues with the poison of the Pfeiffer's bacillus and their reaction to it is a phenomenon which we have to treat in influenza. Miss Valentine's work on our patients has shown that many different strains of influenza bacillus are involved, and Dr Park succinctly sums up the question when he says "the flu" permits the invasion of your own influenza bacillus. This changed condition of the reactivity of the tissues to an irritant is shown in the Schick reaction in one of the patients at this hospital who was very ill with a double bronchopneumonia. The injection of diphtheria toxin on the thirteenth day of her illness was followed by a necrosis of the tissues at the site of the injection. This patient subsequently recovered under the plan of treatment outlined in this lecture.

The local congestion which we have now discussed must be sharply contrasted with a cyanosis which has an entirely different cause. This cyanosis or fuchsia-colored appearance of the lips, seen in so many of these cases, is due to a congestion of the lung and emphysema and the consequent changes in the circulation in the head. This congestion is also present during the asthmatic phenomena of anaphylaxis or serum sickness. These two types of congestion, local and pulmonary, seem to me to be the determinative pathologic condition in the disease as we meet it and to give direction to our therapeutic attempts.

The occurrence of a local congestion is an edema phenomenon associated with a locking of fluid in the tissues. This fact must be kept in mind, for when the swelling state terminates

there may be a sudden release of fluid which may prove disastrous. The characteristic sweatings are evidence of the occurrence of free water. Like many colloidal phenomena the pectinization or coagulation process does not cease at a medium state, but proceeds to such a degree that the patients appear emaciated. This local congestion determines by its localization many of the varying clinical aspects of the disease, and if the disease is viewed from this angle much of its protean character disappears.

Congestion and absorption from the nasal mucous membranes are responsible for the enlargement of the posterior cervical nodes. The local swelling in the nose accounts for the headache and nose-bleed which are such frequent and distressing symptoms. If this swelling involves the uppermost recesses of the nose the drainage may be into the meningeal system and symptoms of a meningism may result. This was shown in a patient, a lad eighteen years of age, who, during the height of the epidemic, suddenly developed a temperature of 105.4° F., severe headache, retraction of the head, Kernig's sign, rigidity of the neck, and somnolence. This boy had obstructed nares, enlarged posterior cervical nodes, and marked deviation of the septum due to maldevelopment of the maxilla. The shrinking effect of adrenalin dropped into his nose caused the entire disappearance of the symptoms in eighteen hours.

At the Willard Parker Hospital it was a noteworthy fact that the only cases of complicating sinusitis occurred in Asians, who have very low nasal bridges and large frontal sinuses, or in men with narrow nasal passages. The use of adrenalin was of distinct benefit in preventing head complications and relieving headache, the zone of central forehead hyperalgesia disappears after adrenalin. The use of aspirin was rarely resorted to. The use of adrenalin had an additional rationale, as it seemed to prevent the absorption of toxin from the nose. Toxin carried in the blood may act in the presence of Pfeiffer's bacillus on the lung cells as a sensitizing agent and assist in the production of the local reaction we term "bronchopneumonia." The anaphylactic nature of this reaction is suggested by the late appearance of the phenomenon, usually six days or more after the head invasion.

Its sudden onset at times with all the phenomena of an acute asthma suggests the validity of applying to the human Manwaring's experiments on guinea-pigs as an explanation of this asthma. The congestion in the lungs most frequently involves the lower lobes.

The profound changes in the tissues induced by the disease seem to be associated with a clinical disturbance of the distribution of the lime in the body. This is manifested not only by the edema or change in the emulsion state, but by the following clinical evidence: changes in the menstruation, changes in the teeth, falling of the hair, the very marked response to stimulation with drugs, such as adrenalin and pituitrin which increases the power of lime utilization by the body, and, finally, evident loss of resistance to disease foci. There is further evidence of the lowered resistance in the greater susceptibility of individuals with lowered lime serum content, such as chronic alcoholics or hyperthyroids and pregnant women. The age incidence of influenza between twenty and forty may be accounted for by the fact that during that period the lime is being utilized in body metabolism.

These profound changes are evidently produced by some toxin. Physicians say that the patients appear "toxic." What they really mean is that they observe the poor perfusion of the capillaries of the face with slight swelling of the tissues. This may be induced by the action of a toxin upon the sympathetic nervous system. The peripheral circulation is poorly maintained during the course of this disease. The French writers, especially Sergent, have emphasized the importance of this phenomenon. When the skin is gently stroked over the abdomen or elsewhere, the stroke is followed by the appearance of a white streak instead of the usual red one of health. I have learned to pay great heed to the prognostic value of the occurrence of Sergent's sign, and it is a great relief to find it disappear after the use of adrenalin or pituitrin, for it is an evidence of the recovery of the sympathetic tone, and I have seen few patients recover where the white line has been persistently present.

The experimental laboratory evidence offered by Miss Parker,

a biologist at the College of Physicians and Surgeons, and Dr Hatfield, of our laboratory, of the existence of a filterable toxin obtained from the Pfeiffer bacillus seems very convincing and should point the way to the production of an antitoxin.

These remarks lead me to a discussion of the methods which the body possesses to destroy such filterable toxins. It is frequently stated that a toxin may be removed from the body by lavage of the tissues. The crowding of fluids for the purpose of lavaging the tissues is a frequent practice. Possibly the toxin may be diluted by such a method, but the bacteria are by no means eliminated, and new toxin is constantly being produced. No intelligent physician would attempt to treat diphtheria, the symptoms of which are also due to a toxin, by crowding fluids. It has been definitely shown that the diphtheria toxin cannot be removed from the body after it is fixed to the tissues.

The disadvantages of lavage in influenza far outweigh the advantages. The tissues of the kidneys are damaged as well as the tissues of the circulatory apparatus, and the introduction of large quantities of a crystalloid (water) imposes a great strain upon both systems. Later in the disease, when the emulsion state in the congested areas is broken and there is a critical release of free water, it may be impossible for all the water to be absorbed and normally circulated. Indeed, the giving of large quantities of water may lead to a retention of the emulsion state with a resulting gelatinous condition of the tissues. Such a case came to autopsy at the Willard Parker Hospital, a man who had successive portions of the lungs involved in sequence. When autopsied on the twenty-eighth day of the disease the entire lungs were gelatinous.

Finally, the giving of large quantities of water causes of itself congestion and increased secretion of the lungs, as anyone who has observed the benefits of water deprivation in the congested lungs of cardiac disease may have observed. I have frequently seen attacks of faintness and coughing one hour after a meal or drink, which in such cardiac patients is equivalent to a stalling of their motor with fluid.

There are still other disadvantages from crowding water

The lavage of the kidneys deprives the body of salts which it can ill afford to lose, and which may deflocuate protein and liberate toxin which had been absorbed earlier in the disease. The mechanism of detoxication in the body is by adsorption, precipitation, and oxidation. The toxemia of influenza may be met by reducing the absorption of toxin from the lung by diminishing the area of infected tissue perfused, by the use of adrenalin in the nose and mouth or subcutaneously, absolute rest in bed, and in certain cases by the performance of a partial pneumothorax by the method of W Parry Morgan.

Precipitation may be accomplished by the introduction of convalescent serum as Redden, of Boston, has employed it, or by the introduction of the intravenous or subcutaneous use of various drugs or substances. At the Willard Parker Hospital I have been observing the results from the subcutaneous use of boiled milk with some apparently successful clinical results, which I attribute to the pectization or coagulation induced. It is possible that the apparent benefit in certain cases, after the early use of salicylates, is due to the coagulation of protein in changing the surface conditions in the blood.

Oxidation is favored by suitable methods in reducing congestion in the respiratory passages, such as the local application or subcutaneous use of adrenalin.

So serious a matter is the excess of free water in the body in influenza patients that it is safe to say it is responsible for many deaths. In fact, the majority of influenza patients die in one of two ways. Some drown in their own secretions, struggling for air, with an intact nervous system, but faulty heart and lungs. This manner of death may occur after the crisis of the disease when the temperature has become normal, and is largely due to the inability of the body to rid itself of water which is suddenly liberated. It should be forestalled by the limitation of fluids. Exception must be made in the case of children who do not stand water deprivation and whose tissues are naturally more turgid than those of adults and have a greater capacity for absorbing water in an emergency. These drowning patients are most pathetic, as they become more and more restless and realize their

inability to breathe. Phlebotomy is of little value in these cases because the removal of blood lowers the blood pressure, especially in the coronary arteries. Deprivation of fluid increases the viscosity of the blood and raises the blood pressure.

It is probably on this account that such marked variations in the prognosis of cases have been noted by many observers from week to week, as this may be conditioned by the gas tension in the tissues, which, in its turn, is the result of barometric conditions. These cases are at times temporarily relieved by the use of atropin and cupping. Pituitrin, which seems to increase the hydration capacity of the tissues, is useful if the amount of water to be taken up is not too great and it is given sufficiently early.

Many dying patients present another picture. These have a paralysis of the vasomotor system. They have a very low blood pressure and their extremities are cyanosed and cold. The blood becomes pooled in the great vessels of the abdomen and frequently the more intelligent ones complain of a substernal anguish. That death is not due to failure of the heart is evidenced by its automatic action for many hours after the patients are unconscious. These patients have had their nervous systems damaged either as a result of blood deprivation or of the toxin. When the vasomotor system has been damaged for a relatively short time it does not recover, so that it is essential that the condition be promptly recognized. This may be done by careful observation of the patient, and I have learned to rely upon the symptom of irritability or wakefulness.

This wakefulness, if it cannot be relieved, is an omen of very sinister import. It may be explained as the result of poor circulation in the brain, or by the primary action of the toxin upon the nervous system. Patients who do not sleep do ill, and although I have seen such patients recover, it is only as a result of successful treatment, for their natural course is downward. Such patients may seem well oriented and apparently rational, but they become wildly delirious suddenly and may leave their beds and so exhaust the circulation. Such patients are the ones who show anxiety in their delirium, as well as cunning, treachery, and suspicion, and frequently assault their attendants. One such

patient, after three sleepless nights and days, started from his bed for the purpose of slaying his wife, whom he suddenly accused of trying to murder him, and when grasped by the attendant, exhausted himself with his struggles so that he died

When the patients have come to this condition no treatment is efficacious. Sleep must be procured, and for this purpose the best drug is morphin, in $\frac{1}{4}$ -grain doses hypodermically. Atropin should not be used in these cases because of its action on the capillaries, not only of the brain but of the skin. It increases the delirium. Occasionally we have used barbital (veronal) in small doses, but it is useless even as a symptomatic remedy in the more advanced cases. The action of morphin probably depends upon its selective action in increasing the utilization of lime by the nervous system. In certain cases the administration of 15-grain doses of precipitated chalk, four times a day, has aided in securing sleep. Some of the patients obtain sleep as a result of hydrotherapy, or from the improved circulation from digitalis. The prompt treatment of insomnia in influenza patients is indicative of a far-seeing and careful physician. Patients who have been somnolent have a better prognosis than those who are wakeful.

It is not my purpose to discuss the various ways in which influenza patients die, as this would require a separate discussion. At times foresight and watchfulness may prevent a tragedy, but unfortunately, as in the cases of singultus (hiccup) or of marked disturbance in the cardiac mechanism, I know of no remedy which will avert death. In the latter case irreversible colloidal changes seem to have been induced.

We have considered thus far the usual symptomatology of influenza involving the head and chest. We have not mentioned the mechanical difficulties associated with the acute pulmonary and tissue emphysema which are closely related as cause and effect. These conditions are amenable to mechanical treatment, the details of which I will discuss more fully at another time.

I have discussed at considerable length the fallacy of forcing fluids in influenza, and I have observed what seems to me an equally atrocious practice in the initial treatment of the disease.

I refer to the use of drastic catharsis, and especially the use of calomel. Mercury has a selective action upon the nervous system, and it seems hardly rational to add a mineral poison in the presence of a vegetable or bacterial toxin. The constipation which so frequently occurs at the onset of influenza is due to over-stimulation of the sympathetic nervous system induced either by the primary invasion of the poison or by pressure from the swelling of the head mucosa. If no cathartic is given, the spasm relaxes and the bowels move. The justice of this view is borne out by the fact that diarrhea occasionally occurs. As the years have passed the catharsis at the Willard Parker Hospital has become less and less drastic in infectious diseases without detriment to the patients, and it is interesting to note that our mortality among our influenza patients was 8 per cent., whereas at a certain naval station, where according to official report calomel was a routine, the mortality was 20 per cent.

In patients whose bowels have moved with the invasion there is no advantage to be gained from purging. A mild laxative may be useful in the other cases. At times, as the result of relief of nasal pressure by the use of adrenalin, the bowels move spontaneously. Violent purging in acute disease is a relic of the practice which obtained when evil spirits were evacuated from the bowels, the larger the movement, the more *noxia* was removed, and therefore the more drastic the purge, the better the practice. The practice of violent purging is on a par with the anti-pyretic treatment of fever.

A recognition of the fact that the influenza poison spends itself on the sympathetic nervous system leads to one other practice in the management of these patients. They should be kept absolutely at rest in bed. Many patients exhaust the sympathetic nervous system by migrations to the bath-room or toilet as a result of purges, and during the epidemic it was a source of frequent comment that many patients fainted on leaving their beds. Several patients were brought into the Willard Parker with scalp wounds sustained in this way, and in one the question of skull fracture was seriously discussed. These patients poisoned vascular nerves and a greater mass of blood in

pumped through the body, and this is best accomplished in the prone position. Even without special nursing, these patients should be provided with urinals and bedpans should be brought to them.

So much for the general handling of these patients. They should be kept in bed, they should not be purged or persuaded that the drinking of much water will rid them of their poisons.

Certain symptoms require special attention.

Headache is best relieved by relieving the congestion through the use of adrenalin dropped into the nose, or by a 4 per cent. antipyrin spray. Adrenalin is preferable because it is absorbed from the mucous membrane and acts as a stimulant. At the Willard Parker, 3 minimis of adrenalin chlorid 1 1000 are dropped into each nostril, the head being sharply extended over the edge of the bed, and 3 minimis placed under the tongue every three hours. This is called the adrenalin treatment. That adrenalin is useful in this way we have ample evidence in the comfort which it gives these patients, and in the improvement in their general condition. If further evidence of absorption were needed I might narrate the case of a diabetic whose sugar tolerance was correctly balanced and in whom 10 drops of adrenalin given under the tongue produced glycosuria.

We have rarely found it necessary to use aspirin, though occasionally 5 grains has seemed to give prompt relief from headache. Occasionally it has been necessary to irrigate the nose. The use of codein or morphin is not indicated for headache alone.

Coughing is another troublesome symptom. This may be due to congestion of the pharynx or even an edema of the uvula. In some cases a spray of 1 per cent benzyl-alcohol emulsion, which we have extensively employed, both anesthetizes the pharynx and seems to have a distinctly healing action on the mucous membrane. The irritation may be further down the respiratory tract. At times there is subglottic swelling, and even edema and redness of the cords, associated with hoarseness. Sometimes the irritation is in the bronchi. Such cases are best relieved by morphin, or by counterirritation with flaxseed poultices or mustard paste applications, or by drugs which dilate

the bronchi, such as *hyoscyamus*, 10 minimis of the tincture. Sometimes there is a pleurisy. Such cases should be relieved by strapping with adhesive plaster so as to fix the border of the ribs.

Opiates should not be used to the point of abolishing the cough reflex, but the cough should be controlled to the extent that it is only productive. The amount of sputum can be very much reduced by limiting the fluid intake, but this should not be carried so far as to permit the sputum to become too tenacious. If this should happen, bicarbonate of soda by mouth is a useful remedy. Anyone who has seen thro' a bronchoscope a mass of sputum thrown from one bronchus to another must realize that coughing itself spreads the infection in the lung and should, therefore, be controlled. Besides, coughing puts a violent strain upon the circulation in the chest.

The usual irritant expectorants should not be employed. There is no reason for giving ammonium chlorid or other expectorants, such as *squill*, as a routine. The mucous membranes are already swollen and irritated. *Squill*, which is present in the ordinary Stokes' expectorant, stimulates the kidneys to excrete water, this is unnecessary if the total fluid intake has been restricted to proper limits. The cough, in the chest cases, is the result of inflammation of the tracheobronchial mucous membrane. Swelling and congestion with subsequent desiccation and exfoliation of the mucous membrane is the pathologic process. For several days the cough is tight, then it becomes loose and productive, and finally disappears even if no drugs are employed, provided the fluid intake has been regulated.

A distressing symptom associated with the cough is pain or tightness in the chest. This may be due to the congestion of the bronchi or to the dilatation of the vessels in the lung. When there is pain, there is usually hyperalgesia, commonly located in the sixth to the eighth dorsal segments or posterior maxima. If the eighth dorsal segment is involved the emphysema reflex may be liberated. If the pain is associated with hyperalgesia, a counterirritant, such as a mustard pack, or cupping, may be employed. Coden or morphin should be employed in these cases early, so as to prevent, if possible, the occurrence of a marked

emphysema. The cases with pleurisy may be relieved by strapping. In one such case where the strapping was ineffectual artificial pneumothorax was induced with prompt relief of pain, immediate fall of temperature, and convalescence.

Dyspnea may be a very trying symptom and may be due to obstruction in the upper air-passages. In some cases tubage of the larynx has been successfully instituted, as recommended by Henry L. Lynah. It is important in these cases to inspect the larynx by the direct vision method of Dr. Lynah lest the obstruction be mistaken for diphtheria. The association, however, of emphysema with a croupy cough and a clear voice are very suggestive of influenzal subglottic involvement.

At times the dyspnea is due to the acute pulmonary emphysema. This may be relieved by counterirritation to the lower portion of the chest, by morphin, and by adrenalin hypodermically administered, in several cases the dyspnea has been relieved by the reduction of increased negative pressure through the performance of a partial artificial pneumothorax.

Very rarely spontaneous pneumothorax is a source of embarrassment if the pressure becomes too high. In these cases the intrathoracic pressure may be reduced by the insertion of a needle.

The most oppressive dyspnea of excessive secretion into the lungs should be anticipated and avoided by the limitation of the fluid ingested. The dyspnea of circulatory weakness must be met by appropriate stimulation.

Occasionally hemoptysis occurs. At times the hemorrhage is massive. In these cases the congestion in the lungs must be reduced by limiting fluids.

Stimulation—So much misunderstanding exists regarding what is suitable stimulation that it seems worth while to restate the problem of stimulation from my own point of view.

What is the purpose of stimulation? To answer this question requires a statement of those conditions which it is sought to maintain in the circulation, namely, an adequate perfusion of tissue with healthy blood. The circulation may fail by reason of failure of the heart, but this is by no means the most important

point of weakness. In an experience extending, during this epidemic, to over 500 pneumonias I have seen only 2 cases in which death was due primarily to cardiac disease. In these cases there was a disturbance of the heart mechanism. The heart pumps powerfully for a long time after the circulation is hopelessly inadequate, so that we must seek the defect elsewhere than in the heart. If that is the case, what is it we do when we say we stimulate the heart? The problem may be restated from the viewpoint of colloid chemistry. We seek to make a colloid mechanism continue to work adequately. The studies of MacDonald and Burridge on the heart muscle contractions in the presence of various so-called cardiac stimulants are of very great importance in this connection. The contraction of muscle is a colloid phenomenon, which may be expressed as a change in dispersity of the colloid particles. During contraction the colloid aggregates increase, in relaxation they are more dispersed. It has been shown that calcium salts are necessary to contraction, while sodium salts are essential to relaxation. All the so-called cardiac stimulants have been experimentally shown by Burridge to have the property of increasing the utilization of lime by the heart muscle. This is not by any means the same as increasing the amount of lime to be used.

The importance of posture must be borne in mind in handling these cases. Colloidal phenomena are surface phenomena, therefore, increase in the surface area of the heart muscle, of dilation of the heart, is favorable for heart action, as has been shown by Starling in his Linacre Lecture on the Law of the Heart. It is not only good muscular action on the part of the heart muscle, however, which must be considered in maintaining the circulation. The circulation may fail in spite of strong cardiac contractions, and this may be induced either by actual reduction of the blood mass or by reason of the blood becoming pooled in the body. In extensive areas of inflammation of the lung a large volume of blood may be collected, much larger because of the negative pressure in the chest than in inflammatory areas elsewhere in the body. The blood may not only pool in the chest, it may pool in capillary beds elsewhere in the body, or in the great

veins of the abdomen. This pooling may be produced as a direct action of a toxin on the capillaries or on the autonomic nervous system, or by failure of the autonomic nervous system in consequence of its impairment or exhaustion.

Morphin—What can we do to maintain the circulation in the presence of the condition outlined above? Probably, in the sense used above, the most important stimulant is morphin. It has a selective action upon the nervous system which biologically and colloidally may be considered a glorified muscle. Many of the phenomena of stimulation and relaxation in muscle are paralleled in the excitation of nerves. Morphin probably increases the utilization of lime by the nerves and assists in the calcification of the synapse. By this action it delays the transmission of impulses through the central nervous system, and in this way prevents the exhaustion of the vasomotor or respiratory centers in response to the painful stimuli from the lungs. It procures sleep. Patients who cannot be made to sleep, die. A $\frac{1}{4}$ -grain of morphin with $\frac{1}{100}$ grain of atropin, repeated if necessary on successive nights, has frequently changed the prognosis of desperately ill cases.

Digitalis increases the utilization of lime by the colloids of the heart. I have already stated my reasons for believing that lime metabolism is disturbed in influenza, 15 minims of a good tincture of digitalis three times a day for several days at a time proves a useful stimulation.

Adrenalin acts like other stimulants by increasing the utilization of lime. This effect is exerted not only on the capillaries of the skin and the lungs, but there is also an effect on the heart. Patterson has shown that the output of blood from the heart is increased by adrenalin, especially in the presence of carbon dioxide poisoning. The adrenalin may be given either through the respiratory or oral mucous membranes, or may be injected subcutaneously, 3 to 5 minims are instilled into each nostril and placed under the tongue every three hours, 15 minims may be given every hour or two, if necessary, by hypodermic.

Pituitrin is the drug which we have found most useful. This we have given in $\frac{1}{2}$ c.c. of the surgical preparation every six hours.

or oftener hypodermically. The effect seems to be manifold. The most important result is an increase in the blood pressure. This is probably due to the general action of the drug on all muscle tissue, increasing the strength of contraction by increasing the utilization of lime. This action seems to be on the capillaries as well as upon the heart and other viscera. Obstetricians have long recognized, sometimes to their sorrow, this tonic action on the uterus.

We have not reserved the use of pituitrin for marked tympanites, for when tympanites develops the nerve centers have been badly damaged. The drug diminishes the output of urine and increases retention of products which may increase the viscosity of the blood. In one case it caused anuria lasting eighteen hours without permanent detriment to the patient, who recovered from a three-lobe involvement.

We have not considered pregnancy a contraindication to the use of pituitrin. Twice we have given it to pregnant women in their eighth month without inducing labor. In one patient in whom the circulation seemed about to fail, 1 c.c. of pituitrin injected hypodermically caused a tonic contraction of the uterus and such a marked improvement in the circulation that no further treatment was required.

We were led to adopt the six hour interval of dosage by our observations in diabetes insipidus, where we noted that the effect of the drug upon the specific gravity of the urine lasted six hours at least.

Alcohol exerts its stimulating action, as Burridge has shown, by increasing the utilization of lime by the muscle colloids just as adrenalin and pituitrin do.

Other Agents—Caffein we have not used because of its irritation of the nervous system, nor have we used camphor, because of its irritating effect upon the subcutaneous tissues. Patients whose nervous system is excited by reason of oxygen deprivation, or exhausted by reason of painful stimuli, should be given morphin by preference. Strychnin in ordinary dosage increases the utilization of lime, but the frequent repetition of a podermic injection does not raise the blood pressure, as

shown by Herrick and, therefore, it is useless to give it for this purpose

We have rarely sought to restore blood volume by saline infusion, and then unsuccessfully, but we have relied upon the contraction of the circulatory bed induced by the drugs mentioned separately, or we have sought to drive the blood from the lungs by decreasing the negative pressure, either by partial artificial pneumothorax or by posture. The recumbent posture which is chosen by most patients increases the cardiac content 20 per cent, dilating the heart, and thus favoring powerful muscular contraction. Sometimes, however, and I have found it an evil omen, patients rise from bed. This seems to be an unconscious effort to diminish the thoracic content and give blood to the general circulation. One of my worst cases was carried through successfully with the aid of a back-rest. It has seemed to me that the marked circulatory prostration following influenza is due in part to the diminution in blood mass as the result of colloid shrinking which goes *pari passu* with body emaciation, and which is restored slowly with improved nutrition. It must not be forgotten that small areas of infection may continue in the lung or sinuses of the head and require diagnostic ability and x-ray assistance for their location. One patient continued weak and miserable for five weeks, when he suddenly collapsed and developed a chill and rise of temperature. Several days later a diagnosable interlobar empyema revealed itself. This man had been frequently examined for the source of his absorption without success.

In conclusion, the whole subject may be summed up by the statement that the phenomena of influenza seem to be the result of profound changes in the swelling state of the colloids of the body in response to an irritant, and from this conception we may build up the clinical picture and deduce the therapeutic indications.

CONTRIBUTION BY DR. WILLIAM H. SHELDON

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THE HOSPITAL AS A HEALTH UNIT

Criticism of Prevailing Methods in Management of Out-patient Departments of Hospitals Districting Large Communities of Value in the Prophylaxis and Cure of Disease in its Early Stages. Importance of Functional Re-education. Value of Social Service Department Necessity for the Study of Practical Problems in Medicine

It has become increasingly obvious as the result of the Great War that closer relations must be established between the practitioners of medicine and the general public. The appalling number of men throughout the country shown by the draft examinations to be physically deficient has driven home both to the medical profession and the public the necessity for a closer co-operation and a more general dissemination of the knowledge of both medical and social factors which tend to prevent disease.

There is a wide-spread sense of social obligation today and a more comprehensive idea of the value of individual life than has ever existed before. The apparent callousness of past times is slowly but surely giving way to a desire to ameliorate certain social and medical conditions which have only been touched upon in the past. Therefore, not only is the medical man expected to diffuse his knowledge in a practical way to the public, but he must also call upon the help of the social workers and others who have the power of assisting him in the effort to bring about the general betterment of mankind.

It would seem that the natural unit for this work would be the hospital. But the hospital up to the present has been almost

entirely given over to the purely medical and surgical treatment of the very sick, with little or no attention paid to the prevention of disease. Such factors as those of occupation, environment, etc., have received scant attention, and the educational possibilities of the hospital with relation to the public have been largely neglected.

The most practical way of making the hospital the important educational center it should be is to district the cities so that there will be one hospital to a given district, and arrangements made that no person applying for medical aid be allowed to go to any hospital outside of that district. The advantage of this method of districting is so obvious that it is surprising that it has not been brought about in the past, though it has been very often advocated.

One of the chief reasons why it has not been done is through lack of understanding on the part of the hospital boards of trustees, the members of which are too often chosen because of their social and political importance and wealth rather than for their keen interest in and knowledge of questions relating to the public health. Moreover, the objection has been made that by so districting the cities there would be an uneven distribution of clinical material for pre- or postgraduate instruction. Anyone who has had experience in clinical teaching can easily combat this argument. It is not a small number of patients which produces a lack of clinical material, but insufficiently thorough methods of examination to determine accurate diagnoses which have been responsible for this criticism. Too often clinicians prefer to pass over a vast amount of material and pick out the obviously sick rather than be more careful in their examination of every patient in order to find cases of disease in its incipiency.

The competition between hospitals has also been a great factor in preventing this districting. Boards of trustees have been prone to point with pride to the large number of patients treated at low cost per patient rather than to records showing a smaller number of patients treated, but with a high percentage of relief and cure.

We should not look so much at the question of the large num-

ber of patients treated as to the welfare of the individual. Therefore, every individual who applies for treatment at the hospital must be carefully examined and his condition diagnosed by all known methods in order to insure proper treatment. That this is done in a large number of in patient departments in the hospitals is, of course, true, but owing to the slipshod and careless methods of the out patient departments it is only the obviously sick that, in a large proportion of cases, ever gain proper care. Owing to the pernicious idea that has existed and still exists in relation to the *numbers* of patients treated, the out patient departments have been exploited in a most disastrous manner, and have been more and more neglected, until at the present time conditions are such that it is difficult to get the graduate of the modern medical school and hospital to work in such a department. For in such a dispensary the doctor is a sort of clerk who sits at a desk and writes prescriptions. He has little time to utilize methods of diagnosis even though they are supplied to him, which is seldom. A large number of the patients seen in this way naturally form a very poor opinion of the practice of medicine, and this growing feeling on the part of the public is of very serious import.

If we are to get the necessary clinical material out of a smaller number of patients, the cost per patient is obviously much greater. But this objection may be obviated by the fact that the economic waste of the present system is tremendous. Why such a large percentage of hospital funds should be spent upon in patients, quite a proportion of whom are hopelessly ill, and so small a part of the funds, if any at all (many hospitals actually making something from out patient departments), appropriated to the out patient department, where a large number of the patients are in such condition that their disease may be either arrested in its incipiency or cured, is one of the mysteries of our medical times. The prophylaxis and cure of disease in its early stages in a large number of persons is of greater economic importance and far greater benefit to the community than the study and relief of a comparatively small number in an advanced state of illness.

If we have the cities districted, as suggested, all sorts of possibilities for good work will be presented. In the first place, such a districting would soon change the point of view of the managers of hospitals. More perfect equipment of the out-patient department would have to be installed, more modern methods of diagnosis adopted, and an adequate staff of doctors provided.

In organizing the staff of the up-to-date dispensary it is essential that the chief of the service should be selected not only because of his known ability in his specialty but also that he should be able as a teacher to instruct his assistants. This is of the greatest importance. Men working in clinics do so primarily to gain knowledge, and frequently, because of the lack of diagnostic facilities in the clinic and insufficiency of time and the inability of the chief to instruct them, they get discouraged and give up the work. Another important point is that every man should receive credit for good work done and his efforts should not result in his work being exploited for the benefit of the chief. As he deserves it, he should receive advancement and opportunities. This also applies in the case of surgical clinics where, as things are organized at present, the chief of the service frequently takes all the interesting operations, and under these circumstances his assistants have little opportunity to gain confidence and operative ability of their own. Taking it for granted that there shall be established an out-patient department on strictly modern lines, comparable to the service within the hospital, there will be almost unlimited opportunities for the extension of work in a given district.

One of the most important considerations of the present day in connection with the welfare of the community is to have every individual thoroughly examined and their difficulties and weaknesses pointed out. This could be accomplished by a sufficient number of well-equipped out-patient clinics. I believe that with a little advertising such institutions would receive the cordial support of the population of each district, were it understood that not only the sick but the well who wished a physical examination could apply and receive adequate attention. Could

we get them so to apply, our facilities for improving public health would be enormous. No one who has worked in out-patient departments for many years can doubt the truth of this assertion. Those of us who aided in conducting the draft and who have been in dispensaries for a number of years have observed an enormous number of defects in individuals which will ultimately become a menace to their health, and possibly their lives, which could be easily rectified had we adequate control of the patients and facilities for correction.

As an example, take the question of the teeth. Roughly, I should say, there is not one person in a hundred among working men and women who does not need dental attention. The condition of the mouths of many is to be observed only with amazement. Pyorrhea in its most advanced stages, obviously abscessed teeth, dental caries, and other of the most gross lesions exist, and at the present time we have few facilities for their correction. Each out-patient department of the city should have an adequate dental department, and when these conditions are found the patient could immediately be referred to this department for correction.

For an example in another field, take neglected and badly treated fractures and injuries, as well as those treated by the most modern methods of ordinary surgery. The number of cripples who have lost function through lack of attention to functional re-education is amazing. I have talked to a large number of corporation physicians, and they all tell the same story. No matter where they send the patient, even to the most approved scientific hospitals, he gets surgical treatment, but not functional restoration. Now in the treatment of soldiers, both abroad and in this country, it has been shown that there are possibilities in functional restoration which were heretofore undreamed of. Unfortunately in America we have only one civil institution as yet for functional re-education, the Clinic for Functional Re-Education in New York City. The equipment for this particular work includes apparatus for hydrotherapy, electrotherapy, thermotherapy, and mechanotherapy. It is a well-known fact that physicians are not practising what they

preach in connection with these methods of therapeutics, but I think there are few who do not thoroughly believe in them. Yet how many hospitals have more than two such departments for this purpose at the most? and yet it has been proved that the use of all these methods is absolutely necessary for the correction of deformities. They are applicable not only to the correction of deformities but also to a large number of patients with nervous and internal disorders. In fact, in a very large group of cases the principal therapeutic reliance must be placed upon these various methods of physiotherapy.

We may also point out what is obvious, that they are of enormous advantage in the treatment of a large variety of medical cases. The cost of installing the appliances necessary for this treatment is very great, but the number of cases referred by casualty insurance companies is so large that such a department, with their co-operation, would be largely self-supporting. Manufacturers are becoming increasingly aware of the fact that they are unable to get restoration of function for their injured employees, and they are becoming anxious to see that such means are provided. Without doubt their cordial support and co-operation would be given to such a department, properly run, in the hospital.

There is a great need in connection with every hospital for the establishment of a gymnasium. Present methods of handling the subject of scientific exercise are much to be deplored. Men who are put into the gymnasiums do largely as they please. Some overexercise, a few underexercise, and the patient is liable to overdevelop certain parts of his body to the detriment of other parts. Very little stress is laid upon the development of chest and abdominal muscles, which, after all, is most needed to insure good health. In gymnasiums the correction of deformities is largely neglected. During the winter of 1917-18, at Cornell Medical College, we conducted a class for rejected men, navy and marine. These men were all unfit, underdeveloped, or had some cardiac functional conditions. Many of these men had been attending Y. M. C. A. and other gymnasiums where they had practically no oversight and had become utterly discouraged from ob-

taining no results. By means of our system of exercises and directions in hygiene and diet a large number overcame their defects, volunteered again, and were accepted. In these classes it was not uncommon for the men to gain from 1 to 3 pounds a week for a number of weeks in the correction of their physical deficiencies, without the use of any apparatus, simply by exercises devoted to the improvement of their specific defect. This small experiment was sufficient to show the economic gain that could be derived from such methods (See preliminary report, New York Medical Journal, October 19, 1918, p 703.)

There are a large number of our hospital patients whom we could refer to a gymnasium with the expectation of ridding them of many of the sources of their ill health. I feel sure that such work could be undertaken with the proper oversight and point of view in mind, but at the present time I know of no public institution in New York City to which a patient can be referred and obtain attention and proper direction for any specific condition. Such a gymnasium need not have elaborate equipment. A well aired room, a few simple pieces of apparatus and mats are all that is required, and this could be run at a very moderate cost. The correction of one class of patients alone would be adequate incentive for the establishment of such a gymnasium. Patients with low-placed stomachs and colons have shown brilliant results from such gymnastic treatment, and yet at the present time our large gastric clinic at Cornell has been unable to find a single public institution where we can refer our patients for proper attention and results. Our knowledge that such results can be obtained is founded upon our experience, as we have had private instructors take charge of the correction of our dispensary patients and produce the given results. Moreover, all doctors know that by referring patients to properly supervised private gymsnasia they are able to obtain the desired results. The use of exercises in cardiac cases also has become of increasing value. During the war thousands of patients with certain cardiac disturbances were treated by such methods by the governments of England, France, and the United States, with gratifying results.

If we had hospital districts in the city with adequately equipped out-patient departments the educational possibilities, in addition to the correction of obvious defects, would be enormous, lectures and talks could be given on various subjects of hygiene, the question of personal health could be emphasized, leaflets with information as to the most approved methods of caring for the general health could be distributed, lectures on the prevention of infectious disease would be of value, industrial hygiene would be of importance, and the public could be brought to realize the significance in the relation of industry to public health

The hours of work which are so much discussed at the present time have largely been left to the careless and indiscriminate decision of labor leaders compromising with the desire of the manufacturer to get the longest number of hours possible from the men. The hours of work are really a medical problem, inasmuch as the health and welfare of the individual are the ultimate reasons for the duration of work, and yet at the present time the medical profession has had very little to say on this all-important subject, largely through lack of realization of how much the health of the individual depends upon the kind of work he does or the number of hours in which he does it. Large corporations are becoming increasingly aware of the desirability of medical opinions on these subjects, and many concerns have a medical staff organized to consider this problem among others. It is interesting to note that with somewhat reduced hours and the same pay many concerns are finding that there is actually an increase in the amount of work accomplished.

It seems as though through a thorough medical study of this subject many of the inequalities and injustices in the demands of both labor and capital could be settled. It is obvious that if an individual works for sufficiently long hours to injure his health the employer is not getting the best benefit from his services. If this could be pointed out by a thorough medical study, many difficulties would be smoothed out. On the other hand, too short hours of work, while theoretically not injuring the health of the individual, in actual practice may do so, it largely depends upon

what the individual is going to do with his hours of leisure, and if these are unduly long the time spent in pool rooms, bars, saloons, and other undesirable places would actually be worse for the health of the individual than if he were at work.

The question of food for the worker could be gone into extensively. Modern medicine contains many phases of scientific knowledge as to the desirability, cost, and value of various articles of food, and yet the general public, in spite of the educational campaign which has been conducted during the war, seems to be largely ignorant of the facts which have become common knowledge in medicine.

Information concerning these matters of health and hygiene could be conveyed to visitors to the clinics by means of printed leaflets. Cornell University Medical College, I believe, was almost the pioneer in the printing of slips for clinic patients. Some nine or ten years ago, with the cordial support of W. Gilman Thompson, I wrote a series of slips¹ which we have been distributing ever since. These include instructions for patients with nephritis, heart disease, diabetes, constipation, gastric ulcer, chronic gastritis, hypochlorhydria, lead-poisoning, chronic joint disease, and chronic bronchitis. The slips give the patient definite rules to follow as to his daily routine, food and habits, and the precautions necessary for him to observe to combat his disease. It is explained why it is necessary for him to follow these rules and the benefit that will accrue to him if he does so. There is a detailed diet list for each group of cases. There is also a leaflet on the general care of health, and a pamphlet on diet, with balanced menus and rules for cooking.

To indicate the prevailing interest in this subject, we have been asked by scores of corporations, such as insurance companies, colleges, and hospitals throughout the country, for several hundreds of sheets of these printed slips for distribution, and have even had requests from medical schools in foreign countries for such slips, and permission to translate them. We have been increasingly impressed with the value of such slips. Not only is good done to the patient, but very often the health of the whole

¹ See Printed Slips for Clinic Patients, Medical Record

family is improved by his newly acquired knowledge of hygienic matters. An extensive campaign of printed slips in any district would surely be of enormous value.

In order to accomplish all that is desired in the scheme which is here outlined an extensive department of social service would be necessary. Medicine in the past has too often stood away from the practical problems, as I think I have indicated, and it is generally accepted now that the health of the individual is more than a matter of germs or the ordinary considerations which we take up in medicine. It is the outcome of the combination of all the circumstances which surround the individual—the place, method of living, wages, working place, the number of children in the family, etc.—all those elements which we know enter into the problem of the individual. This becomes increasingly important at the present time during a stage of economic unrest. In a hospital district such as we propose it would be necessary to have the social service workers investigate not only the conditions which we have mentioned but also help in the problem of education.

For example, if a man comes to the out-patient department and has bad teeth, it should be the duty of the social service to follow up that man until his mouth condition is cured. If a man is suffering from psychasthenia, it should be the duty of the social service to investigate his home to see whether his surroundings are compatible with his mental health. Too often we try to cure by medicine instead of curing social conditions the difficulties of which are really overwhelming the individual. Perhaps it may be a low wage with a large number of children, or insufficient food, or little prospect for the future.

I believe that an adequate social service could do much toward remedying the problem of infectious diseases. While this is a function essentially of the boards of health, their data is so lacking in correlation as to districts, and conditions of living are so inadequately followed up that it is far from satisfactory, and one cannot form any very accurate deductions from their statistics. If all the sick poor were from a certain district or if most of them were reporting to a given hospital, it would be quite

social service department to gather accurate statistics as to the prevalence of certain diseases in certain sections, just as tuberculosis district clinics have been of enormous value in collection of such evidence. If in a certain tenement there is a number of cases of typhoid, that tenement could be investigated as to the presence of carriers or other sources of infection, the cases eliminated. We can cite example after example here. Certain blocks in a district would be found to be really unhealthful. The social service could find out the causes which caused this. Unsanitary conditions of tenements, defiance to tenement laws, and other causes of misery could be pointed out, and reform made easier.

Moreover, there is no reason why such a social service could not be extended, or even the question of employment considered. Every section of all great cities has cripples and other handicapped individuals who are thoroughly discouraged and need help to obtain work that is suitable for them. That such an idea is capable of practical application is being shown at the Clinic for Functional Re-Education, where placement is a kind of social service. At this clinic the possibilities of the individual are carefully investigated, his physical handicaps noted, he is brought into contact with various agencies distributed throughout this city and other cities and encouraged in every possible way to adopt some employment which is suitable to him. Not only that, but connected with the social service there is a board of advisors made up of men prominent in various lines of endeavor, who are willing to take their time to consult with these individuals and advise them as to the opportunities and advancement in their special line of work.

The social service as related to occupational conditions has an importance which can hardly be exaggerated. In the case of chronic cardiac disease there is a great need of putting these people in proper vocations. As it is, a large number of cardiac patients either become chronic invalids or do work that is unsuited to their condition. It is a very pitiful condition of affairs when a man has to pursue an industry which in the course of months to several years will surely kill him, and where

facilities for his readjustment are so slight. By co-operating with such institutions as now exist, and should exist were our system established, institutions such as the Bureau of the Handicapped, more extensive study could be made of cardiac patients in order to understand better what positions in industry they are fitted to fill and those they are not fitted to fill

The same reasoning applies to tubercular patients, but that subject is fairly well taken care of by present organizations and board of health clinics

By the investigation of patients coming to hospitals evidence is soon found of the relation of industry to disease, and by a large number of such observations and investigations classifications can be made as to how the individual reacts to certain types of work. An interesting example of this was demonstrated a few years ago. A certain street railway, with the kindest intentions, supplied the motormen with stools, but the stools were all of the same height. A social worker observed that in the Cornell Clinic there were a number of men suffering from sciatica who were employed by this company. On investigation it was found that owing to the standard stool the taller individuals got a continuous pressure on the sciatic nerve through ringing their gong with the right foot, which brought on a neuralgia. The attention of the company was called to the defect and stools of varying height were supplied. In this way probably hundreds of cases of disability were avoided by the company and considerable misery escaped by a number of motormen.

Cases of lead-poisoning came to our attention in a dozen individuals, foreigners who could not speak English, who were found to come from a certain factory. Investigation showed that in this factory, which was run largely by foreign labor, warnings as to the dangers in the manufacture of white lead were only printed in English, and therefore, while the few English employees were not acquiring cases of lead-poisoning, a large number of the foreigners were. When this was pointed out to the manufacturer the head of the factory had the warnings printed in various languages, and the number of cases of lead-poisoning was enormously diminished.

Cases have been discovered by the social service and called to our attention where workers in various trades, through lack of knowledge on their and the manufacturer's part, were seriously affected by bronchitis through respiration of dust. By instructing these individuals in how to guard against this by wearing face-masks the dangers were largely done away with.

There is no reason why such a social service could not go into the problem of occupational therapy. A large number of people coming to hospitals are not physically fitted for their regular work. These could be trained in basket making, rug making, typewriting, bookkeeping, and other occupations suited to their handicaps. At the Chnic for Functional Re Education the Social Service Department has worked out an entirely practical solution of the difficulties of many men.

Through such a social service as is proposed it would be possible to help people to find recreation, for, as is well known, healthful recreation is one of the solutions of many medical and social problems. Information could be given as to free lectures, free dances, moving pictures, etc., and the patients encouraged to go to the museums and other public places of interest, and in this way be aided in getting normal recreation. It would be a great advantage to bring the patients needing relaxation into contact with the various forms of wholesome pleasure which exist, for often they are unaware of even the greater organizations of our country, such as the Y M C A., Knights of Columbus, and others who are working for the betterment and happiness of the individual.

There seems to be every reason why there should be closer co-operation between the public schools and such a district hospital as is here outlined. The hospital physicians could be examining physicians of the schools, and as defects are discovered in the students, they could be referred to the hospital where there would be adequate facilities for correcting these defects, and not, as is so often the case now, defects diagnosed and yet not corrected. Does it not seem that this could be of enormous advantage to the community at large and an economical arrangement of the school and city authorities? This also has the ad-

vantage of removing the school physician from politics, and the fact that the examining physician is subject to the authority of a well-organized hospital would be sufficient indication of his ability and honesty. The city could well afford from the economic standpoint to pay the hospital for such services rendered both as to examination and as to correction of defects.

CLINIC OF DR. A. S. BLUMGARTIN

LENOX HILL HOSPITAL

CASES ILLUSTRATING DIAGNOSTIC PROBLEMS

I. Primary Malignant Tumor of the Lung II Cerebro-spinal Syphilis III Three Cases Illustrating Problems in Nephritis (a) A Problem in Diagnosis (b) Problem in Treatment (c) A Problem in Prognosis and the Effect of Decapsulation. IV Two Cases Illustrating the Diagnosis of Aortic Syphilis (a) Early Aortic Syphilis (b) Aneurysm of the Arch of the Aorta.

I. PRIMARY MALIGNANT TUMOR OF THE LUNG

R. H., a machinist, thirty seven years of age, married, was admitted to the Lenox Hill Hospital complaining of sore throat, pain in the left side of the chest, slight hoarseness, and loss in weight.

history that might be of diagnostic value other than the loss of weight and a very remote family history of pulmonary tuberculosis. Indeed, the patient was sent into the hospital from the dispensary with a diagnosis of pulmonary tuberculosis.

Physical examination shows a middle-aged man, quite anemic, and suffering only from hoarseness which has grown progressively worse while he has been under observation. The chest is normal.



Fig. 222.—Roentgenogram of chest of R. H., showing mediastinal mass in case of primary malignant lung tumor.

in shape. There is an area of dulness over the mediastinum about twice the normal width, extending from the suprasternal notch to the area of cardiac dulness in the anterior left chest. Here there are signs of a large amount of fluid, which on repeated puncture proved to be serosanguineous in character. There are signs of a small amount of fluid at the base of the right chest posteriorly (Figs. 222-224).

Examination of the abdomen is negative. Examination of the extremities shows no evidence of any lesion.

The urine examination was negative. The sputum has been examined a number of times, but no tubercle bacilli were found. The blood pressure was 110 systolic and 80 diastolic. The Wassermann examination of the blood was negative. The Wassermann examination done again ten days after a provocative sal-



Fig. 223.—Roentgenogram of chest of R. H. showing increase in size of mediastinal mass while patient was under observation and mass in lung. Case of primary malignant tumor of lung.

varsan injection was also negative. The Wassermann test of the spinal fluid was negative. The blood count shows evidence of secondary anemia with 75 per cent of hemoglobin. About five days after admission 10 c.c. of a serosanguineous fluid was removed from the left chest and carefully examined for evidences of tumor cells but none were found. On culture, this fluid was sterile.

The only evidence, therefore, of a pathologic lesion in this case is in the chest

The presence of a serosanguineous pleural fluid at once indicated the necessity of an $\text{\textit{r}}$ -ray examination of the chest, and the roentgenograms reveal the presence of a mediastinal mass involving the entire mediastinum from the suprasternal notch to the cardiac shadow. The mass itself is irregular in outline and did not pulsate on several fluoroscopic examinations.

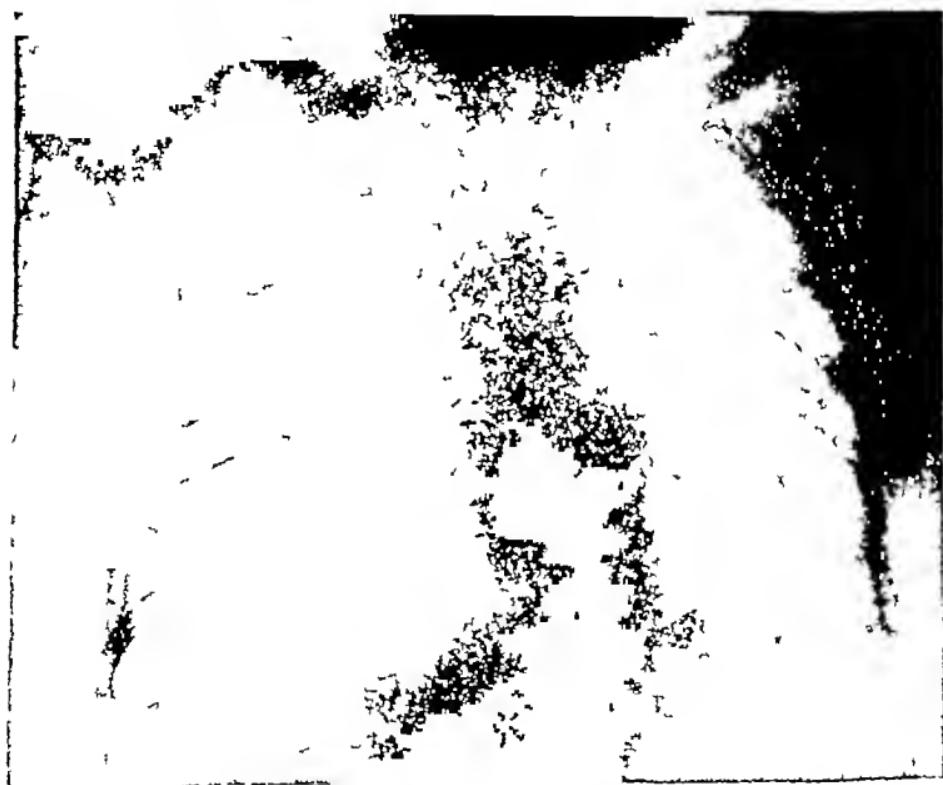


Fig 224.—Roentgenogram of chest of R. H., showing mediastinal mass and definite mass in lung. Case of primary malignant tumor of lung.

Examination of the larynx showed a paralysis of the left recurrent laryngeal nerve, and repeated examinations of the larynx showed that this paralysis became complete while the patient was under observation.

Gradually we noticed that an inequality of the pupils developed, the left being smaller than the right. (This may have

been due either to pressure with almost complete paralysis of

the left sympathetic nerve, or to pressure with irritation of the right.) Both pupils show a diminution in reaction to both light and accommodation.

The case, therefore, presents the possibility of the following lesions, with their inherent difficulties in differential diagnosis: 1, Mediastinal tumor, 2, aneurysm of the arch of the aorta, 3, lung tumor.

The occurrence of a recurrent laryngeal paralysis would seem to point to a mediastinal lesion. The pulmonary symptoms, however, were present for a long time before the occurrence of hoarseness, the symptom which actually brought the patient to the hospital. Furthermore, a large amount of serosanguineous fluid is present in the left chest which precludes the determination of a mass in the left lung by physical examination. In spite of the fact that the presence of a mass cannot be shown in the left lung even by the *x* ray examination, the occurrence of persistent pulmonary symptoms on the left side for such a long time, together with the presence of a large amount of serosanguineous fluid in the left chest, would still suggest a lesion in the left lung. The involvement in the left recurrent laryngeal and sympathetic nerves very late in the course of the disease increases the probability of a lung tumor as the most logical diagnosis. If it is a lung tumor, it is probably malignant, and we can explain the finding of the mediastinal mass as a secondary metastasis.

We may exclude the presence of an aneurysm by the absence of the physical signs of an aneurysm, by the lack of pulsation in the fluoroscopic examinations of the mediastinal mass, by the absence of a syphilitic history, and by the negative Wassermann test in both blood and spinal fluid. We must bear in mind, however, that an aneurysm in the arch of the aorta is the most common mediastinal tumor encountered.

About three weeks after admission we first felt in the left suprACLAVICULAR space a rather flattened nodular mass about the size of a walnut which seemed to be firmly adherent to the apex of the lung. This was probably a metastatic suprACLAVICULAR carcinomatous lymph node.

A few days later a bronchoscopic examination was done by

Dr Felix Cohn, who found the following lesion. In the region of the trachea about the bifurcation but above the larynx was seen a stenosis. The lumen was elliptical, the long diameter being from the left anteriorly to the right posteriorly, and it seemed to be due to a right-sided pressure. Its structure was soft, as the bronchoscope easily dilated it and passed it. The lumen was filled with mucus and was in itself quite small, making it very difficult to see the tracheal bifurcation. The vocal cords were apparently involved similarly, as they did not move on respiration.

Although study of the roentgenograms shows the presence of a mediastinal mass with radiations into the hilus of both lungs, and accordingly we could be tempted to diagnose a primary mediastinal tumor with secondary involvement of the lung, this does not correspond to the clinical history of the disease. The clinical course and the presence of a left supraclavicular node would seem to indicate that the case is one of primary lung tumor with secondary involvement of the mediastinum rather than a primary mediastinal tumor with secondary lung involvement. The study of the later x-ray pictures, taken several days after the first ones, seems to show a more definite lung involvement on the left side. To correlate this finding with the clinical course, it is probable that the tumor may have originated in the region of the hilus. The history of marked cachexia and the occurrence of metastases and a serosanguineous fluid in the chest all point to the tumor being a malignant one, probably a sarcoma or carcinoma. Such a tumor would be more apt to originate in the region of the hilus rather than near the pleural surface, which is the common site for endotheliomata and which produces cachexia late in the course of the disease.

We should, therefore, consider the case as a primary malignant tumor of the left lung, originating in the region of the hilus and producing metastases in the mediastinum and left supraclavicular node. We cannot say whether it is a sarcoma or a carcinoma, although sarcoma is the more frequent tumor in an individual of the age of this patient.

Note—Subsequent to the above the patient died about one

one week after his admission to the hospital. An autopsy performed by Dr. G. H. Rohdenburg showed a neoplasm of the lung with extension into the mediastinum. The following findings were the only important pathologic lesions:

The pleural cavity of the left side was obliterated by dense adhesions to the parietal wall. That of the right side was divided by numerous firm adhesions. On removing the larynx, lungs, and heart *in toto* the following condition was seen. Behind the trachea and almost completely encircling it was an irregular, lobulated, firm mass, yellowish white in color, which extended forward over the aorta from the supraclavicular notch to the base of the heart. Posteriorly it extended from the level of the tracheal notch to the diaphragm and laterally for a distance of 10 cm. to the left and 5 cm. to the right of the aorta. This mass was a direct continuation of a circular encapsulated mass in the upper posterior portion of the upper lobe of the left lung. The mass in the lung measured 7 cm. in diameter.

On section, the growth showed a striated appearance and a milky juice could be expressed from it. The neoplasm completely encircled and obstructed the lumen of the trachea at the bifurcation.

The right lung showed two small areas of healed tuberculosis at the apex, but was otherwise normal. There were no gross metastases in either lung.

The heart and pericardium were normal.

Microscopic examination of the tumor showed it to consist of cells, in general packed closely together and showing no special arrangement, although there were areas in which the cells were collected into islands or columns or formed the lining of alveolar spaces. The nuclei of the cells were round or ovoid, rich in protoplasm, and filling up the greater part of the cell. Mitotic figures were fairly abundant. The stroma varied in amount, but was relatively scanty. Fibrils separating the individual cells could not be demonstrated.

Anatomic diagnosis Sarcoma

II CEREBROSPINAL SYPHILIS

M K, a widow, forty-seven years of age, was admitted to the Lenox Hill Hospital complaining of pain in the lower abdomen for the past week which was made worse by eating. The illness began with cramp-like pain in the lower abdomen. The pain did not radiate. She took an emetic a few days before admission, which did not relieve the pain. About this time she had gaseous eructations after eating and a sour taste in the mouth. She had chills at the onset of this illness and again the day before admission. She had not eaten anything for three or four days because of lack of appetite and because food gave her pain. The bowels had not moved for a week, nor did she pass any urine during that time until the day before admission. There was no past history of any definite condition other than an attack of rheumatism eight years ago, at which time her wrists and ankles were swollen, but she was not confined to bed very long. She had a pubiotomy done at childbirth.

Ordinarily, the patient does her own housework. She drinks an occasional cup of coffee and her appetite is usually good. She does not take alcohol in any form. Her bowels move regularly and she urinates about five times daily and twice at night. She sleeps well.

Menstruation began at the age of seventeen and was always regular until the age of twenty-six, when her first and only child was born. Following this birth she had amenorrhea for only eight weeks, when she began to menstruate again very profusely for about two weeks, and this was followed by a foul leukorrhea for some time. Since that time she has never menstruated and there is no history of miscarriage.

The patient's father died of erysipelas at the age of sixty and her mother of dropsy at the age of seventy-two. She has one brother and one sister, both of whom are living and well.

Physical examination shows a small, poorly nourished woman of middle age, but who looks older than she really is. She is of almost dwarf-like stature. There is a slight pigmented eruption about the chest, front and back, and both the skin and mucous membranes show evidence of marked anemia.

The extremities show only an absence of knee-jerks. There is no edema and no evidence of any lesion. Vaginal and rectal examinations were negative.

Eye examination by Dr. Diem showed an incipient cataract in the left eye and a left ophthalmoplegia *externa* and *interna*. The fundus of the right eye was normal.

Careful examination of the nervous system showed only the following positive findings: 1, Sluggish mentality, 2, left ophthalmoplegia *externa* and *interna*, inequality of the pupils both of which are irregular, the left being wider than the right, an Argyll-Robertson, an external strabismus of the left eye, and a ptosis of the left eye, 3, hyperesthesia of the lower extremities, 4, absence of knee-jerks, 5, paresis of the bladder and colon.

Urine examination on admission gave an alkaline reaction, specific gravity 1021, trace of albumin, 0.3 per cent. sugar, trace of acetone, many leukocytes, a few urate crystals, and many bacteria. Two weeks later, examination of the urine showed it to be very turbid and to contain very many pus-cells and many red blood-cells, including a few small blood-clots.

Examination of the gastric contents showed free acid of 38, total acidity 62, a suggestive lactic acid test, and blood by the guaiac test. Microscopically, a few partly digested starch granules and fat globules could be seen.

Chemical examination of the blood showed a CO₂ combining power of 57 and blood-sugar per cent 0.076.

Roentgenographic examination of the genito-urinary tract taken five days after admission failed to reveal any calculi in the kidneys or in any part of the urinary tract. Roentgenographic examination of the stomach showed it to be normal in size and with no evidence of ulcer. There was, however, some six-hour retention, which was probably due to spasm. There is also an appearance of spasm at the cardia which is quite marked. Fluoroscopic examination eliminated the possibility of a malignant condition of the lower end of the esophagus and proved it to be a cardiospasm.

Blood examination done about six days after admission showed red blood-cells 3,970,000, white blood-cells 5600, poly-

nuclears 77 per cent., small lymphocytes 14 per cent., mononuclears 5 per cent., transitionals 3 per cent., eosinophils 1 per cent., hemoglobin 75 per cent. The clotting time of the blood was four minutes. The blood pressure taken five days after admission was systolic 130, diastolic 85. The blood Wassermann done about six days after admission was 4 plus. Twenty days after admission a lumbar puncture was done and 20 c.c. of a clear fluid was withdrawn under considerable pressure. Examination of this fluid showed a cell count of 75, globulin of 3 to 4 plus, and a 10 plus Wassermann. Colloidal gold test done on this fluid was unreliable.

This history in a woman of the age of this patient would give the impression of a gastric or hepatic lesion, probably malignant, or of a renal lesion. From the sudden onset of the symptoms, however, we might possibly suspect a crisis in the course of a nervous condition. The only factor in the history that might be of etiologic value is the fact that at the age of twenty-six, eight weeks after the birth of her child, she developed a persistent uterine discharge, and she has never menstruated since nor has she had any other children. It is possible that at that time or a short time previously a luetic infection might have occurred. The only data with reference to the child born at that time is the fact that he is now twenty-one years of age, and that he voluntarily entered the army and later deserted. (We might possibly consider this a stigma of low mentality.)

On examination we find an old dwarf like woman who seems to be quite emaciated. Her mentality is decidedly sluggish. Examination of her chest shows only a marked scoliosis with convexity to the right and the signs of a chronically depressed right lung, as well as an old, evidently rheumatic valvular lesion. Examination of her abdomen shows a hyperesthetic abdominal wall, with tenderness on pressure in the right upper and lower quadrants, and evidence of a distended bladder and a distended and tender ascending colon.

Examination of her nervous system shows a decidedly diminished mentality, unequal and irregular pupils, the left being wider than the right, but both giving an Argyll-Robertson reaction.

There is also a ptosis of the lid and external strabismus, as well as cataract in the left eye

The history in this case is very misleading, giving us no clue to her neurologic condition. It is that of an abdominal condition with an etiologic factor that might be considered a source of a luetic infection. The physical findings, however, seem to point to a lesion in the nervous system involving areas widely separated, namely, the third nerve nucleus in the corpora quadrigemina and the lower lumbar segments of the cord. Because of the marked divergence of the areas involved the lesions are evidently produced by a vascular disturbance, probably an endarteritis.

Since syphilis is the most common cause for such a lesion in a woman of the age of this patient, and since we can determine a probable definite source of infection in the history of the case, this evidence points to a cerebrospinal lues, in which the lesion is an endarteritis involving the vessels of the third nerve nucleus and the lower lumbar segments of the cord. The Wassermann reaction in the blood increases our suspicion as to the nature of the infection, and the Wassermann reaction of the spinal fluid corroborates the syphilitic origin of the condition.

Now that we have determined the case to be one of cerebrospinal lues, let us attempt to determine the nature of the pathologic lesion. There are three possible pathologic processes: 1, Syphilitic basilar meningitis, 2, syphilitic endarteritis of the brain and cord, 3, gummatous infiltration of the brain.

We may eliminate a syphilitic basilar meningitis from the fact that there are no signs of meningitis present, there is no rigidity of the neck, no Kernig's sign, no Babinski, the patient does not complain of headache nor does she vomit. Furthermore, if a syphilitic basilar meningitis were present, other cranial nerves besides the third would be involved, but since the only nerve involved is the third, the lesion in the brain must be in the third nerve nucleus.

A gummatous infiltration may be eliminated from the fact that even the involvement of a small area of brain tissue would give much more extensive signs than those present, nor would

two widely separated lesions of such a character occur simultaneously

The presence of an endarteritis may be established by the apparent chronicity of the condition, by the evident smallness of the area of nerve tissue involved, by the widely separated areas involved, and by the fact that this is the most common syphilitic lesion of the nervous system in an old person. The evidence pointing to a lesion in the lower lumbar segment of the cord is the hyperesthesia in the lower extremities, the absent knee jerks, and the evident interference with the lower lumbar reflex arcs, as is shown by the paresis of the bladder and colon.

The symptoms for which the patient was admitted to the hospital, however, were those of an acute abdominal lesion. There was no evidence of any lesion of any of the abdominal viscera until roentgenographic examination was made, which showed spasm of the cardia. From this fact, and the fact that the symptoms occurred suddenly, we might consider the abdominal symptoms as those of a gastric crisis occurring in the course of a cerebrospinal lues.

About eighteen days after admission we could feel a distinct tender mass on pressure over the right hypochondrium, and repeated careful examination of this mass showed that it was probably a kidney. The x-ray examination of the genito-urinary tract showed no evidence of stones. From the urinary findings, in addition to the findings on manual examination, we should consider this mass as a pyelonephritis of the right kidney, evidently an ascending infection from a cystitis associated with paresis of the bladder. It is quite probable that the original attack of an acute abdominal lesion for which the patient came to the hospital might have been produced by this kidney lesion.

III. THREE CASES ILLUSTRATING PROBLEMS IN NEPHRITIS

(a) Illustrating a Problem in Diagnosis—J. H., a theater clerk, twenty five years of age, was admitted to the hospital suffering from generalized edema. Eight weeks before admission he noticed that his face, legs, penis, and scrotum had become markedly swollen. The edema did not disappear on lying down.

He also complained of severe headache at times, and had periods when he was quite drowsy, but he had no vomiting and no nausea. The edema of the eyes and face decreased somewhat during the day. He had dyspnea on exertion, but no cough. He had marked polyuria, voiding every two hours during the day and four times at night, and passed about one quart of urine in twenty-four hours. At times the urine was cloudy and he noticed blood in it. He had excessive thirst.

He has been in the hospital twice previously for a similar condition, the first time about a year ago, when he was admitted with a similar history occurring a few days after an excessive alcoholic bout and exposure to cold. The second time was about eight months ago, when he was in the hospital for about two weeks.

He had measles and pertussis as a child and he has had a number of attacks of gonorrhea, but he denies lues. There is no history of an infectious disease. A few years ago he had pulmonary tuberculosis and was in Asheville, North Carolina, for six to eight months, returning unimproved. He was quite alcoholic until eleven months ago, and he smokes excessively, consuming about twenty cigarettes a day.

This history is that of an acute nephritis occurring about one year ago, which improved under hospital treatment and has now recurred. There is no definite etiology for the condition except a New Year's celebration with exposure to cold a week previous to the onset of the original attack. There is no history of an infectious disease, but the patient has had pulmonary tuberculosis, which leads to a suspicion of a surgical lesion of the kidney.

Physical examination shows a well-nourished but decidedly anemic young man, with puffiness under the eyes and generalized edema. His chest shows no signs beyond evidence of a small healed lesion of tuberculosis at the left apex, and examination of the heart is negative. Examination of the abdomen gives no evidence of any lesion except slight edema of the wall.

The urinary output varies from 900 to 1600 c.c. in twenty-four hours, which is usually more than his daily intake of fluids. The urine, which is always distinctly cloudy, shows the pres-

ence of albumen, with a great many pus-cells and blood-cells on microscopic examination. He has a slight urethral discharge, several smears of which show a great many gonococci. On only one occasion, after numerous examinations, was an occasional hyaline cast found. His urea elimination, his ammonia nitrogen, urea nitrogen, and chlorid elimination are normal.

The blood pressure is 160 systolic, 105 diastolic. The blood count shows red blood-cells 1,176,000, normal in appearance, hemoglobin 75 per cent., white blood-cells 13,000, polynuclears 65 per cent., lymphocytes 37 per cent., large mononuclears 1 per cent., and eosinophils 0. The patient ran no temperature while in the hospital. Repeated chemical examinations of the blood showed no evidence of retention of non protein nitrogenous products. The figures average as follows:

Urea nitrogen	12.0
Creatinin	2.0
Uric acid	3.7
Sugar percentage	0.136

Repeated phenolphthalein tests always showed a normal elimination.

Examination of the eye grounds were quite negative. Roentgenographic examination of the urinary tract showed no evidence of calculi.

Although the general appearance of the patient strongly suggests a chronic nephritis, the only evidence of such a lesion is the albuminuria and slightly increased blood pressure. The albuminuria, however, can readily be accounted for by the presence of pus in the urine, especially in the absence of casts. The function of the kidneys seems to be fairly normal. The presence of blood and pus in the urine would seem to indicate that the patient may be suffering from an infection of the genito-urinary tract, possibly situated in the kidney, but exact localization of the lesion cannot be determined until patient can be cystoscoped.

The history of tuberculosis and the presence of an old healed tuberculous lesion of the left lung also leads to a suspicion of a tuberculosis of the kidney, but this does not seem quite as prob-

able as a gonorrhreal condition, because the urine is decidedly purulent and many gonococci are present

The patient was kept in the hospital and his evident gonorrhea treated so as to enable the use of a cystoscope, but he left the hospital before his condition cleared up so that cystoscopy could be done

The interesting feature of this case is the fact that the general appearance of the patient is that of a classical chronic nephritis. But the laboratory examination of the urine and blood show no evidence of a disturbed function of the kidneys nor is there any appreciable rise in the blood-pressure. Indeed, the only evidence of nephritis is the generalized edema. It is quite probable that a surgical condition of the kidney might account for this generalized edema, but while we strongly suspect it, on account of being unable to carry out cystoscopic examination we cannot positively assert that such is the case. Furthermore, a surgical condition of the kidney might also give appreciable evidence of disturbed kidney function. We are dealing, therefore, with a case of generalized edema. There is no evidence of a cardiac lesion, there is no evidence of a lesion of the blood, and there is no evidence of the kidney to account for this. On close questioning we find also that the edema is especially marked in cold weather, and disappears almost entirely in warm weather and in warm climates. Furthermore, the edema is entirely unaffected by the diet, whether it is rich or poor in salt. We are, therefore, dealing with a case of either (1) generalized angioneurotic edema, (2) a surgical lesion of one or both kidneys, or (3) chronic nephritis in such an early stage as not to give any evidence of functional disturbance of the kidney.

(b) Illustrating a Problem in the Treatment of Chronic Nephritis—*S D*, a widower, sixty-two years of age, a salesman by occupation, was admitted to the Lenox Hill Hospital complaining of frequent urination and dyspnea. The illness began four months ago with dyspnea and cough, with mucoid expectoration. For some time previous to the onset he had frequency of urination, but this has increased markedly, until at the present time he has

to get up about every two hours to urinate during the night. He can barely hold his water. Three months ago he began to have edema of the lower extremities and puffiness of the eyelids, which is gradually increasing. For the last year and a half his vision has been bad and has gradually been growing worse daily. At the present time it is so bad that he cannot read a newspaper. For the last week he has had severe headaches. He has spells of weakness, so that he cannot get up out of bed, at such times he is unable to walk, falling down when he attempts to do so, and then he is unable to get up again. He has lost about 40 pounds in weight in the last four weeks. About five months ago he had a hemiparesis. He had diabetes four years ago with carbuncles as a complication. He had measles, typhoid, and pneumonia as a child, and heart and kidney trouble about a year and a half ago. He denies lues. One brother died of apoplexy and another of intestinal tuberculosis.

Examination gives the impression of a considerable loss in weight. There is no evidence of general glandular enlargement. The head shows no evidence of any lesions except for the tortuous temporal vessels. The eyes show pallor of the conjunctiva, the pupils are equal and react both to light and accommodation, the fundi are pale and show a progressive optic atrophy with slightly tortuous vessels, there is no evidence of an albuminuric or diabetic retinitis. There are no evidences of any lesions of the nose, throat, or ears. The tongue is coated and the teeth are in very poor condition.

The chest is barrel shaped and the lungs show the characteristic emphysematous breathing with occasional sibilant rales. The heart is enlarged, the apex-beat being in the sixth interspace 12.5 cm to the left of the median line. The heart sounds are regular, rhythmic, but faint and distant. There is a suggestion of a distant faint systolic murmur to be heard at the apex, which is not transmitted however. The pulse is regular, full and bounding, and there is evidence of arteriosclerosis in the palpable vessels. The blood pressure is 145 systolic and 80 diastolic.

The abdominal wall is soft and flabby. The liver border is felt about three fingerbreadths below the costal margin and is

tender on pressure. The spleen is not palpable. There is no evidence of free fluid in the abdomen. No abnormal masses can be felt. There is a scrotal hernia on the right side. The extremities show no evidence of any lesions. The knee-jerks are absent on both sides. There is evidence of an old right hemiplegia. Rectal examination shows a markedly enlarged prostate. The Wassermann reaction is negative. The daily urinary output varies from 800 to 1300 c.c., which is usually about the same amount as the total fluid intake.

The urine examination always shows a specific gravity varying from 1015 to 1021, a faint trace of albumen, no sugar, and microscopically many pus-cells and occasional hyaline and granular casts. The phenolphthalein tests average from 25 to 48 per cent. The blood examination shows a non-protein nitrogen retention, as the following figures indicate:

Urea nitrogen	83.0
Creatinin	3.7
Uric acid	3.7
Sugar percentage	0.192

The history is that of a cardiac syndrome either primary or secondary to a kidney lesion. There is no definite etiologic factor in the history that indicates whether the heart or kidney was the most probable primary lesion. The marked frequency of urination suggests a lesion of the prostate, and the occurrence of a mild attack of hemiplegia suggests a vascular lesion.

The physical examination shows a rather well-nourished but anemic and stupid old man, who gives evidence of a chronic nephritis, an arteriosclerosis, and a secondary myocardial insufficiency. He also has an old hemiplegia on the right side and quite a large prostate. It is difficult to find any definite etiology for his lesions, but it would seem that the old age and the typhoid and pneumonia are the most important factors. The lesion from which the patient is evidently suffering is situated in the kidneys, and it is evidently a chronic nephritis.

We therefore attempted to determine what was the functional disturbance of the kidney. There is no evidence of any edema,

consequently there is no salt retention. The daily urinary output is normal, consequently there is no water retention. The chemical examination of the blood shows evidence of a retention of non-protein nitrogenous products, as is shown by the increase in the urea, creatinin, and uric acid percentages. The dull, lethargic, stupid mentality may be considered evidence of a mild uremic state which is associated with the non protein nitrogen retention.

The case therefore presents a problem in treatment. What we have to treat is the nitrogen retention. This we can do by eliminating the proteins from the diet so as to lessen the formation of protein waste products, and increasing at the same time all the excretions of all his excretory organs. Instead of reducing the proteins in his diet, by gradually eliminating the protein foods we attempted to determine the amount of proteins the patient could stand without inducing retention, at the same time maintaining his caloric needs. This was done in a manner similar to the Allen treatment of diabetes. The patient was starved for three days, receiving only 16 c.c. of whisky every four hours, and coffee and lemonade up to 500 c.c. daily. The only medication was sodium bicarbonate, 0.6 gram, three times a day. The lemonade and sodium bicarbonate were given for the purpose of preventing a possible acidosis which the starvation might produce. At the end of the three days' starvation another chemical examination of the blood was done. This showed a marked reduction of the nitrogen retention, as the following figures indicate.

Urea nitrogen	20.8
Creatinin	1.85
Uric acid	2.0
Sugar percentage	0.126

The patient was then put on a milk-diet of 500 c.c. for about three days. This contained about 35 grams of protein. A chemical examination of the blood was again done, and it showed no increase in the previous figures.

We then added spinach, baked potatoes, toast, broth, ice-

cream, and orangeade, so that the patient was getting about 60 grams of proteins, about 1960 c c of fluid, and the caloric value of this diet was about 1640. This was kept up for about a week, when a chemical examination of the blood was done again, but it showed no increase in the last figures. We then added string beans and increased the milk to 750 c c, raising the proteins to 80 grams, the fluid to about 2500 c c, and the calories to about 2200.

The only medication during this time was sodium bicarbonate, 0.6 gram, three times a day, magnesium sulphate 30 grams every morning to increase the elimination by the intestinal tract, and tincture of digitalis 0.6 mil for the dyspnea and to improve the circulation in the kidney.

At the end of this time another chemical examination of the blood was done, but it showed no evidence of nitrogen retention.

The patient gradually improved, his stupid, dull, lethargic state disappeared, he gained in weight. This diet was kept up for about two months, the patient finally being discharged improved. His final blood condition showed no evidence of nitrogen retention. His urinary condition, however, remained the same, albumen and casts still being present, but his phenolphthalein tests showed a higher functional ability of the kidneys.

This case is interesting because it is a type of chronic nephritis in which the main functional disturbance in the kidney is in its inability to eliminate nitrogenous waste products, and because we were able to improve the condition by determining the protein tolerance and keeping the patient on such a diet. The patient was suffering from a mild uremia. The uremic state cleared up when the nitrogen retention was reduced by the elimination and subsequent reduction of the proteins in the diet. The tolerance for proteins, which was higher than the normal average requirements, was determined by beginning with starvation and then gradually increasing the amounts while maintaining his caloric needs.

It is interesting to note that the albumen and casts in this case have no relation whatever to the amount of protein intake.

It seems that they are merely evidences of a nephritis, but they do not indicate anything in regard to the ability of the kidney to eliminate protein waste products

(c) Illustrating a Problem in Prognosis and the Effect of Decapsulation—M. M., a carpenter, thirty-six years of age, born in Ireland, married, was admitted to the Lenox Hill Hospital complaining of swelling of the arms, legs, abdomen, and puffiness of the face for a week previous to admission. There was slight dyspnea, but no disturbance of vision and no vomiting. About one year ago he had a similar attack, in which the dyspnea on exertion was more marked, and, in addition, he had failing vision, polyuria, and frequent urination. He continued under treatment for the year following this attack. He has never had any other illness except for frequent attacks of tonsillitis. His occupation made it necessary for him to be out in the cold in the open constantly and he has been a heavy drinker, taking 5 glasses of whisky a day and about 5 glasses of beer. He denies gonorrhea and syphilis. There is nothing relevant in his family history except that his father died of dropsy.

The patient on admission presented the appearance of a fairly well nourished but quite anemic middle aged man, with puffiness of the face and typical generalized edema which suggested at once chronic nephritis. There was nothing abnormal in the head or any of the cranial cavities until the eyes were examined, when it was found that although the reaction to light and accommodation were both normal, he had a slight external strabismus and a nystagmus. The chest was normal and there was no evidence of any lesion in the lungs. The heart, however, was enlarged to the extent that the left border extended about two fingerbreadths to the left of the left nipple line, and the apex beat was heard best in the sixth interspace in the left nipple line. The heart sounds were of poor muscular quality, though they were normal in rate and rhythm. There were no murmurs to be heard, but the second aortic sound was rather faint and muffled.

The abdomen showed no evidence of any lesion and the contained viscera were found to be normal. The blood-pressure

varied from 190 to 210 systolic, with a diastolic of 108 to 116. There was no evidence of arteriosclerosis of the palpable vessels.

The urinary output was within normal limits, varying from 1000 to 1800 c.c. in twenty-four hours, which was usually more than the daily amount of fluid intake. The urine varied, at times being acid and at other times being alkaline in reaction, its specific gravity averaged between 1009 and 1017, it usually contained large amounts of albumen, never any sugar, at times many hyaline and granular casts, and always more or less pus-cells. The phenolphthalein test showed only a faint trace on every examination.

The chemical blood examinations showed a non-protein nitrogen retention, the urea varying from 50 to 63, the creatinin from 7 to 9, the uric acid about 4 to 4.5, the sugar percentage 0.146, and the cholesterol from 150 to 152. The Wassermann reaction of the blood was negative.

This history is that of a classical chronic nephritis which came on suddenly about a year ago. The only factors in the history that we might consider of etiologic value are the occupation, which constantly exposed the patient to cold, and the very marked history of alcoholism. The positive findings all point to the kidneys as the seat of the primary lesion. The case is evidently that of a chronic nephritis with indications of a non-protein nitrogen retention and marked anasarca. In other words, the kidney is unable to eliminate protein waste products and salts. The water elimination seems to be fairly good.

The important symptoms to be treated in this case were the nitrogen retention and the salt retention. Since the evidence of nitrogen retention was very marked, this was the most important condition to treat because the patient's outcome depended largely on this, as uremia was threatening. His edema did not inconvenience him very much. Furthermore, the edema was a simple matter to eliminate, either by limitation of fluids or by a salt-free diet. An attempt, therefore, was made to treat the nitrogen retention by determining his protein tolerance in the same manner as in the previous case. But even when the patient was on starvation for about five or six days the chem-

ysical examination of the blood showed no diminution of his retention, the only result was a loss in weight and a slight diminution in the creatinin from 9 to 7. We increased the protein intake by giving the patient milk and overfeeding with carbohydrates, allowing a diet containing about 60 grams of protein, but this had no appreciable effect upon the nitrogen retention, so that we abandoned this method of treatment and determined to treat the patient rather as an individual, our object being only to make him comfortable, not limiting his diet in any way, and increasing the excretion through all the excretory channels by saline purgatives and by digitalis. The edema gradually subsided and the patient felt better. Consequently, we came to the conclusion that this was a case of chronic nephritis with a persistent disturbance in the function of the kidney so that it was unable to eliminate protein waste products and salts.

The prognosis, therefore, according to the very high non protein nitrogen figures was very dubious. The patient was doomed to suffer from persistent disability of his kidney, and in all probability would not live longer than a year or two. The persistent high figures of creatinin in the blood corroborated this assumption.

We therefore decided to attempt to do a decapsulation of the patient's kidneys and give him the benefit of whatever good effect this operation would have. Consequently, one month after admission, the patient was transferred to the surgical service of Dr. Herman Fischer, and the following day a decapsulation of both kidneys was performed under ether anesthesia. The kidneys at operation were seen to be typically small and contracted.

The patient made an uneventful recovery from the operation. His urinary findings after the operation did not vary from those before, nor did any of the subsequent phenolphthalein tests show any increase in the functional capacity of the kidneys. A week after operation the blood findings were as follows:

Urea nitrogen	77.0
Creatinin	9.0
Uric acid	4.4
Sugar percentage	0.168
CO ₂ combining power	50.0

In other words, the nitrogen retention in the blood was not changed. Twelve days later another blood examination showed

Urea nitrogen	50.0	.
Creatinin	5.4	
Uric acid	3.3	
Sugar percentage	0.152	

This shows a slight improvement in the function of nitrogen elimination, as is indicated by the slight diminution of the creatinin elimination.

The patient gradually improved, his edema subsided, and he left the hospital about three and a half weeks after the operation feeling very comfortable, but with evidences of a nitrogen retention in the blood and a consequent failure to eliminate protein waste products.

This case is interesting because it shows that with persistent high blood figures of nitrogen retention, especially the high creatinin figures, the prognosis is bad because the nitrogen retention cannot be changed, and we cannot hope to gain much from regulation of diet. The patient should merely be made comfortable and any contingency should be treated as it arises. If uremia occurs, this must be treated in the usual way, if a myocardial insufficiency occurs, this should be taken care of. As far as the ultimate outcome is concerned, little is to be expected. The decapsulation operation in this case did not result in any appreciable improvement in the ability of the kidneys to eliminate protein waste products.

IV TWO CASES ILLUSTRATING THE DIAGNOSIS OF AORTIC SYPHILIS

(a) Early Aortic Syphilis — L. M., a widowed coachman, sixty-three years of age, was admitted to the Lenox Hill Hospital complaining chiefly of swelling of the legs of two weeks' duration.

The illness began about two weeks before admission, when he began to notice that he was short of breath and that his legs and abdomen were gradually getting swollen. At the same time he

would cough at night and suffer from severe frontal headache. He has attacks of precordial pain which radiate up to his left shoulder and down the left arm. These attacks have occurred even for some time before the onset of the present illness. Of late his vision has been blurred and he has had marked frequency of urination during the day, and he has had to get up about five or six times a night to urinate. He has lost about 75 pounds in weight during the last few years.

He had measles as a child, as well as frequent attacks of sore throat and joint pains. For the past eight years he has been suffering from heart and kidney trouble. He denies lues but he had gonorrhea about forty years ago.

He is a man of good habits, except that he drinks a glass of beer occasionally and smokes several pipes of tobacco daily and an occasional cigar.

The history is that of a cardiac syndrome, either primary or secondary. A primary vascular lesion of the heart is strongly suggested by the frequent anginal attacks. There is no definite etiologic factor to account for his condition except possibly the rather indefinite history of sore throat and joint pains.

The patient is a well nourished old man, who is slightly dyspneic on lying down. There is no evidence of any lesion until the chest is examined. Here the dulness, diminished breathing, and the distant fine subcrepitant rales at both bases of the lungs behind especially on the left side, strongly suggest the presence of fluid. The heart is enlarged the left border extending to about a fingerbreadth to the left of the left nipple line. A diastolic murmur may be heard at the base, and it is transmitted upward to the left side of the neck and downward and to the left of the apex.

The abdomen shows no evidence of fluid or any lesion. The liver border, however, may be felt indistinctly about three finger breadths below the costal margin. There is no edema of the scrotum or lower extremities. The blood pressure was 165 systolic and 50 diastolic. The Wassermann reaction of the blood is 4 plus. The urine analysis is negative. The phenolphthalein test shows 27 per cent. The chemical examination of the blood

shows no evidence of nitrogen retention. The roentgenographic examination of the chest shows only evidence of an enlarged heart.

The clinical evidence is that of an aortic regurgitation with anginal attacks on a syphilitic basis. The positive evidence of syphilis, in addition to the attacks of pain, evidently anginal in character, proves the presence of a syphilitic aortitis. In spite of the negative x-ray evidence of a dilatation of the aorta this lesion has evidently produced a sufficient dilatation of the aortic ring so that a regurgitation through the semilunar valves is produced. This valvular lesion is now decompensated and the patient is suffering from a myocardial insufficiency.

As regards treatment, the patient should receive intensive treatment with mercury and iodids for about six to eight weeks, and then salvarsan may be given. We never know the exact nature of the pathology of the aortic lesion. Consequently, it is not wise to give salvarsan even when there is a remote possibility of breaking down a syphilitic lesion in the aorta, which might be a fresh one. When salvarsan is given in such cases it should be given in very small quantities of fluid so as to avoid producing a sudden rise in blood-pressure.

(b) *Aneurysm of the Arch of the Aorta* — E. A., a plasterer, sixty-three years of age, unmarried, was admitted to the Lenox Hill Hospital complaining of shortness of breath and swelling of the legs and scrotum for three months.

The illness began about a year ago, when he gradually began to have shortness of breath on slight exertion. At times he would have edema of the legs and face in the evening. He has had to be confined to bed for a few weeks six times in the course of the last year. He now comes to the hospital on account of shortness of breath, edema of the extremities, and cough at night.

At the age of three he suffered from eye trouble, the principal symptoms of which were "spots before the eyes." In a few years the symptoms disappeared. He has never had scarlet fever. Although he has been having attacks of sore throat

every winter, he has never had an attack of rheumatism. He had a gonorrhea when he was twenty, and two years later he had a hard chancre followed by a rash.

Except for moderate beer drinking, averaging about 6 glasses a day, and one or two pipes of tobacco daily, he is a man of fairly good habits.

His father died of stomach trouble at the age of fifty. Besides this there is nothing relevant in his family history.

The history of this patient is apparently that of a myocardial insufficiency probably the result of a primary or secondary heart lesion. From the fact that a definite history of lues is present a primary lesion in the aorta should be strongly suspected.

The patient is a rather plethoric, heavily built man with a characteristic rosacea of the face, and suffering from severe dyspnea. He is markedly cyanotic. There is a generalized edema of the lower extremities, abdominal and thoracic wall extending upward to the level of the scapula.

His chest is barrel shaped and gives a hyperresonant note all over except at the left base behind where the decided dulness diminished fremitus, and bronchial breathing strongly suggest fluid. The mediastinal area of dulness is widened to about twice its normal width. It merges with the cardiac area of dulness in an obtuse angle on both sides. The area of cardiac dulness is widened. Its border extends on the left to the anterior axillary line, where, in the fifth interspace, the apex beat may be heard. The right border extends to the right to about two fingerbreadths to the right of the sternum and obliterating the cardiohepatic angle. The heart sounds can be heard best in the fourth and fifth interspaces around the left nipple, but they are distant and muffled. No murmurs can be heard anywhere (Figs. 225, 226).

The pulse is small and evidently paradoxic in character, and there is distinct evidence of arteriosclerosis in the palpable vessels.

The abdomen is distended, the abdominal wall is edematous, and free fluid is present in the abdominal cavity. The liver border may be felt about three fingerbreadths below the costal

margin, but its consistency is normal. The lower extremities are edematous and old healed ulcers are present in both legs.

The daily urinary output is normal, and the urine has a specific gravity of about 1027 to 1030, and always contains a faint trace of albumen, no sugar, and at times many small hyaline casts.



Fig 225.—Roentgenogram (anteroposterior view) of chest of E. A., showing aneurysm of arch of aorta, pleural and pericardial effusion. Case of aortic syphilis with aneurysm of arch of aorta.

A phenolphthalein test (catherized specimen) could not be done because of a markedly tortuous urethra. The chemical examination of the blood, however, showed only a slight retention of uric acid.

The systolic blood-pressure was 145 and the diastolic 95. The Wassermann test of the blood was negative.

The roentgenographic examination of the chest showed an aneurysm of the beginning of the arch of the aorta, and a very large heart shadow, with possible evidence of fluid in the pericardium and in the left chest.

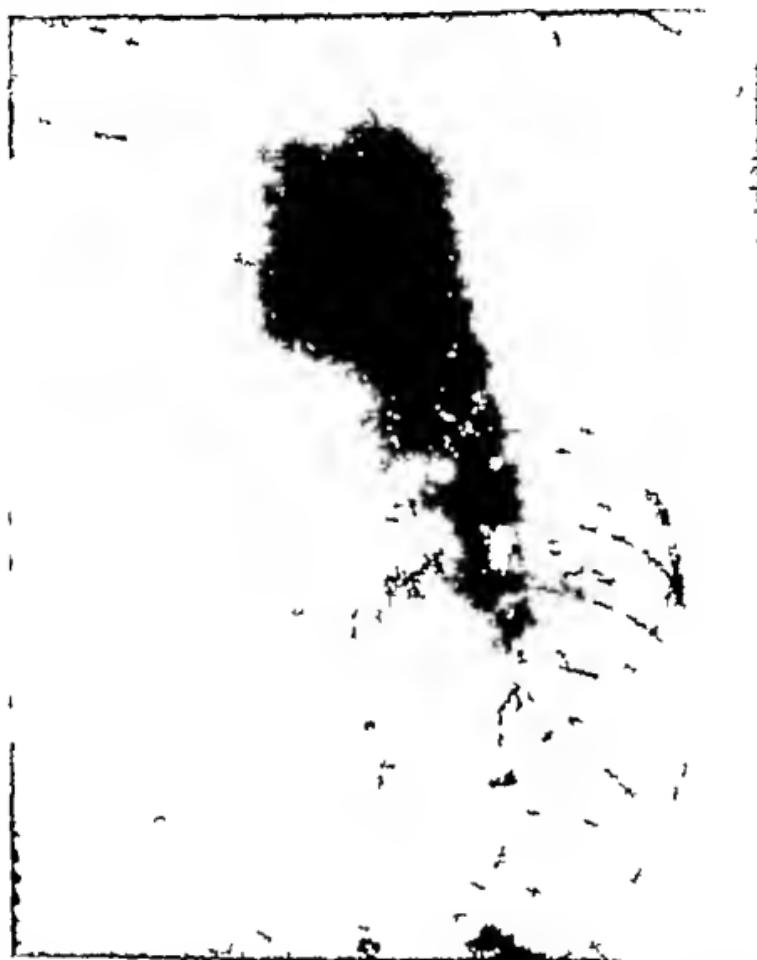


Fig. 226.—Roentgenogram of chest of E. A., showing lateral view of aneurysm of arch of aorta. Case of aortic syphilis with aneurysm of arch of aorta.

The very severe dyspnea and cyanosis and pleural effusion, together with the generalized edema, are evidences of a myocardial insufficiency. The cardiac signs, however, are those of a pericardial effusion, which is evidently secondary to an aneurysm of the arch of the aorta. The evidence pointing to an aneurysm of the arch of the aorta is the widened area of cardiac dulness

and the evidence of the roentgenogram. The Wassermann reaction is negative, but the definite clinical history of lues is just as valuable in determining the etiology as the Wassermann test.

It is interesting to note that the usual physical signs of aneurysm of the arch of the aorta are absent. This is due to the presence of a pericardial effusion, the emphysema of the chest, and to the location of the aneurysm at the very origin of the aorta, as is shown by the roentgenogram. It is an interesting speculation to decide whether the pericardial effusion is due to the aneurysm or to the myocardial insufficiency. While we have no evidence to decide this point, we can, however, logically explain the effusion by the dilated aortic ring pressing on the coronary veins with a consequent stasis and pleural effusion.

CLINIC OF DR. A. McL. STRONG

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AURICULAR TACHYCARDIA IN CHILDREN 2 CASES

THE paroxysmal tachycardias have thus far defied exact etiologic classification. Indeed, it is doubtful if they are susceptible of etiologic classification except in a manner too uncertain to be of practical value, for the functional and the organic appear to be inextricably bound together in their causation.

The usual criteria of true paroxysmal tachycardia are as follows. A very rapid and usually, though not always, regular ventricular rate, abrupt in its onset, and frequently accompanied by the subjective sensation of palpitation. The rate is seldom less than 160, and is often as high as 300 or more. The termination of a paroxysm is characteristically abrupt, and may be followed immediately by a pause similar to the compensatory pause seen after a single premature contraction.

There are four types of abnormal rhythm which may be responsible for the paroxysm. They are auricular tachycardia, auricular flutter, auricular fibrillation, and ventricular tachycardia. At present we are concerned only with the first three. Of these, the first two are the disorders most frequently associated with paroxysmal tachycardia. With the more extensive use of the string galvanometer, however, it has become evident that auricular fibrillation occurs in paroxysms far more frequently than was formerly supposed.

In a large proportion of cases these rhythms are associated with disease of the myocardium, and yet case reports are not lacking to show that any one of them may have a functional or a toxic origin. Hence we are unable to state, in a given case, from the rhythm alone, whether or not organic disease of the heart is

present. In the last analysis our decision in regard to the heart does not rest with the rhythm any more than it rests with a murmur in a case suspected of valvular disease. Always we are compelled to seek the final answer in other data, such as the age of the individual, his habits, past or present, infections, the frequency and duration of the paroxysms, the size and form of the heart, the condition of the valves, of the arteries, the blood-pressure, the adequacy of the peripheral circulation, the state of other organs, as the kidney and the thyroid gland.

The three types of tachycardia mentioned as having their point of stimulus formation in the auricle have this in common—they are all ectopic rhythms. The cardiac pacemaker is no longer situated in the sino-auricular node, but in some other region of the auricular musculature which, by virtue of increased irritability or superior rhythmicity, has vicariously assumed the function of pacemaker for the time being, or even permanently.

In auricular tachycardia the heart responds to stimuli from a single ectopic pacemaker in the auricle, and the ventricle responds to every auricular impulse. That the rhythm is an ectopic one may be demonstrated by the string galvanometer. The P deflection of auricular activity assumes an abnormal form until the paroxysm terminates. The auricular rate is usually less rapid than in auricular flutter, and in the last-named rhythm the ventricle does not respond to every auricular impulse.

The 2 cases about to be described belong to the category of auricular tachycardia, and demonstrate two contrasting clinical types of this condition. Both were under ten years of age. In neither case was it possible to determine with certainty the etiology of the disorder. In one case there was a tendency to spontaneous recovery, apparently with permanent restoration of the normal rhythm. In the other there was not only no tendency to spontaneous return of the normal rhythm, but the tachycardia resisted stubbornly all therapeutic measures, was eventually complicated by evidences of myocardial insufficiency, and ultimately contributed to the fatal outcome of an acute infection. In general, it may be said that the tachycardias of this class are more prone to resist all efforts to slow the heart than are those

extreme borders 37 cm to the right, 53 cm to the left, total transverse diameter, 9 cm

Leukocytes, 16,000, polynuclears, 74 per cent, lymph, 34 per cent, eosinophils, 1 per cent, transitionals, 1 per cent Wassermann reaction negative

Course — The child received no medication throughout her stay in the hospital except one dose of castor oil on admission. Her temperature fell to normal on the second day and remained so throughout. She was put on soft diet and kept in bed for

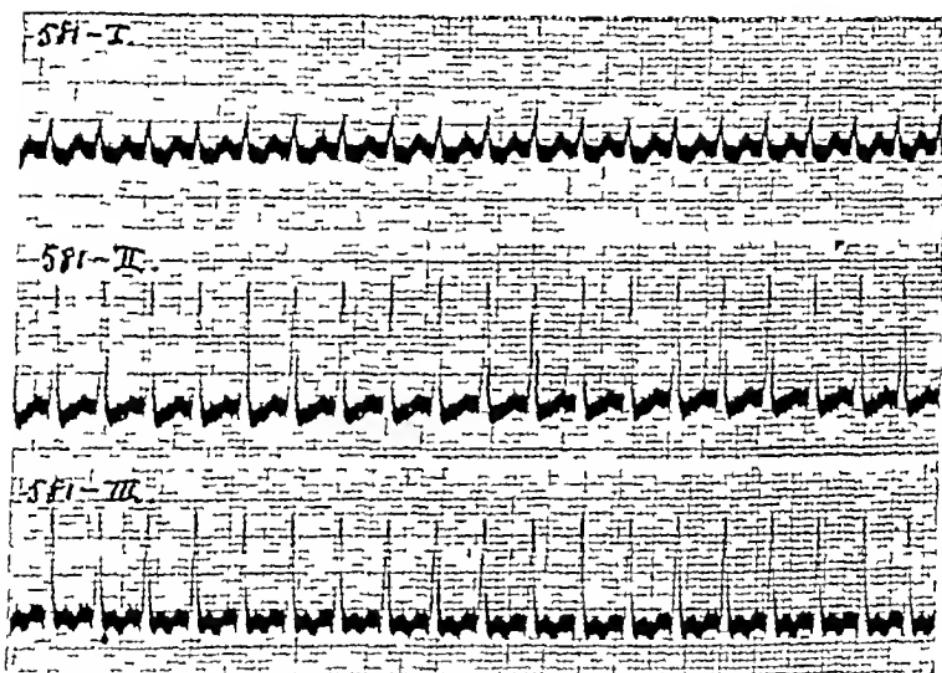


Fig 227.—Electrocardiogram of Case I during the period of tachycardia

several days. On the second day her pulse was 148, on the third day, 78, fourth day, 82, and on the tenth day, just before her discharge from the hospital, 90. The heart action remained perfectly regular throughout.

Over a period of seven months after she was discharged from the hospital the child made twenty-four visits to the dispensary for observation. Usually she admitted some constipation, and once had a sore throat, but usually there were no complaints of any kind. During the first month after leaving the hospital

she had noticed a few short periods of rapid heart action. The ~~last~~ visit was on February 13 1919. She was perfectly well and weighed 50 pounds.

The Electrocardiograms Fig. 227 (taken on day of admission)—The heart rate is 221. The P wave is difficult to identify with certainty, apparently it is inverted in Lead III. The P-R interval as estimated from the apparent foot-point is about 0.14 second. Although the inverted P in Lead III is not enough alone to justify the assumption that the rhythm is an ectopic

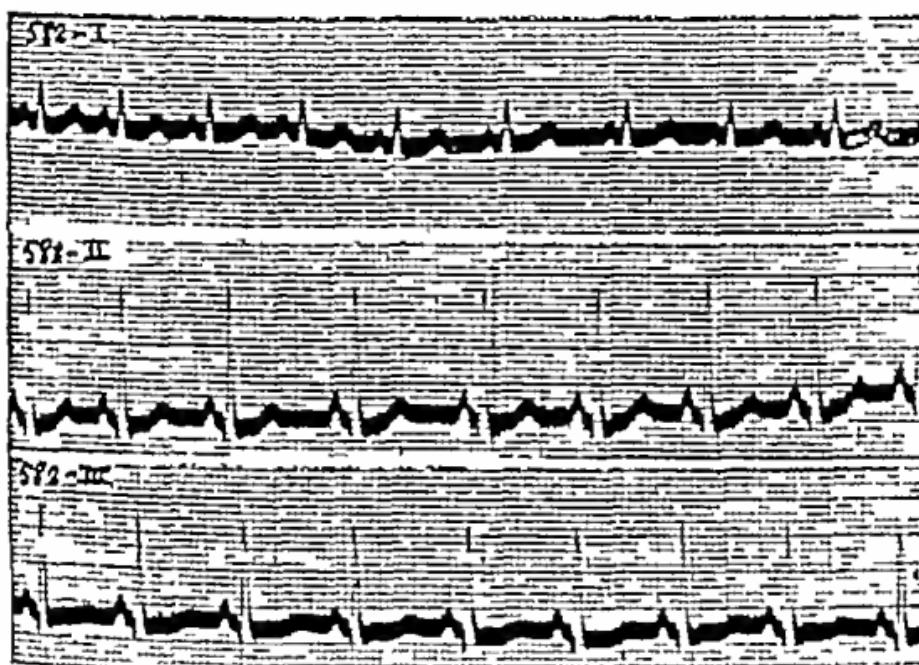


Fig. 228.—Electrocardiogram of Case I after the resumption of the normal sinus rhythm.

one, the record is, in appearance, very characteristic of auricular tachycardia and the rate itself speaks very strongly for this interpretation.

Figure 228, taken on the second day, shows reversion to the normal sinus rhythm. The rate is 88. The P wave is upright throughout, and the P-R interval measures 0.12 second. This record establishes the fact that the previous rhythm was of ectopic origin and belonged in the class of paroxysmal tachycardia commonly known as auricular tachycardia.

Discussion—In this case we have no positive evidence of organic disease of the heart. To state categorically that the heart is normal, however, would be to assume a knowledge which we do not yet possess. It is by watching such individuals for as long a time after the attack as possible that we may eventually learn the significance of the disordered rhythm.

CASE II—A schoolgirl aged five years. Normal birth and feeding, normal development. Home conditions good.

Family History—Father alcoholic, said to suffer from rheumatism. Mother had one miscarriage at two months. Two other children living and well.

Past History—Chickenpox three and a half years ago. Has had German measles. Always subject to colds, which are invariably accompanied by sore throat. During the past year and a half has had three severe attacks of follicular tonsillitis. No rheumatic fever, no joint pains.

Present Illness—About seven weeks ago, following play-hour at school, the teacher noticed that the child looked pale and ill. The school physician said the child had a "rapid heart" and advised sending her to a dispensary. From the dispensary she was sent home, with some medicine, and directed to remain in bed. She did so for three weeks, but the pulse continued at 170 to 180 day and night. It never fell below 150. For the next four weeks the child was up and about, playing as usual, but was very easily fatigued, especially after climbing stairs. The mother thought she was dizzy at times. No headache, no nausea or vomiting, appetite and bowels normal, no pain. She herself did not seem to be conscious of the rapid heart action.

Physical Examination—A pale, well-developed, fairly well-nourished child, without dyspnea or evidence of any discomfort. She walked into the hospital ward. **Mouth** Four carious teeth, tonsils large and red. **Neck** A few small cervical and submaxillary nodes palpable. **Chest** Outwardly normal. **Lungs** clear. **Heart** Right border 3 cm., left border 7.5 cm. from the mid-sternal line. Point of maximum impulse in the fifth space, mid-clavicular line, diffuse in character. Action regular, rapid (180). The sounds of good quality, no murmurs or accentuations.

Teleroentgenogram Length of heart, 11.5 cm Extreme right border 3.5 cm, extreme left border 7.3 cm from midline, total transverse diameter 10.8 cm *Abdomen* Liver palpable at costal margin, spleen not felt. No tenderness present. *Extremities* normal *Reflexes* normal *Temperature* 98.6° F, *respirations* 44, later 28 A throat culture was negative for Klebs-Löffler

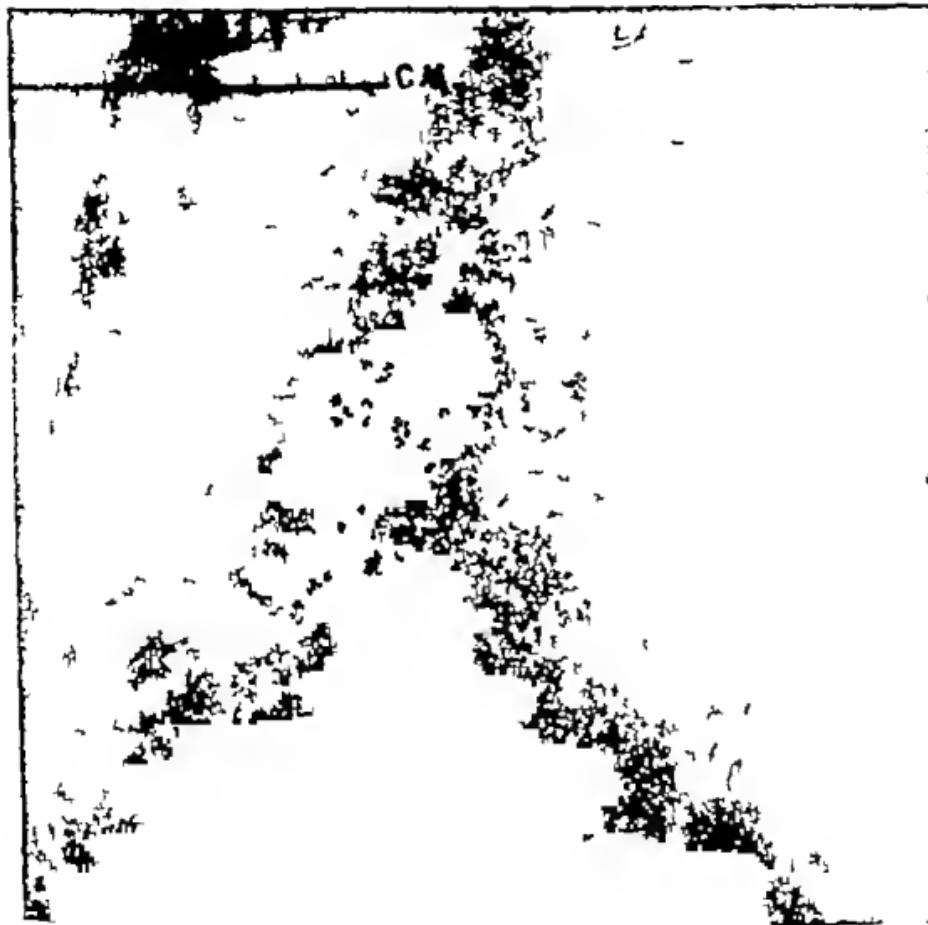


Fig 229.—Teleroentgenogram of Case I

Leukocytes, 11,200, polynuclears, 51 per cent., lymphocytes, 47 per cent., transitionals, 2 per cent Urine was normal (Fig 229)

Course—On the second day the pulse was once found to be 92, a little later 180 On the third day it was 192, always regular That night, while sleeping 145

The patient was given soft diet and kept as quiet as possible

On the ninth day after admission tincture of digitalis was begun in the hope of slowing the heart. It was given for fifteen days in gradually increasing doses, beginning with 6 minims on the first day. By the end of fifteen days she had received approximately the equivalent of 1 gm. of the powdered leaves. At this point the ventricular rate became a little slower and irregular, due to partial heart-block (digitalis). Vagus pressure had been shown to exercise the same effect. The auricular rate showed

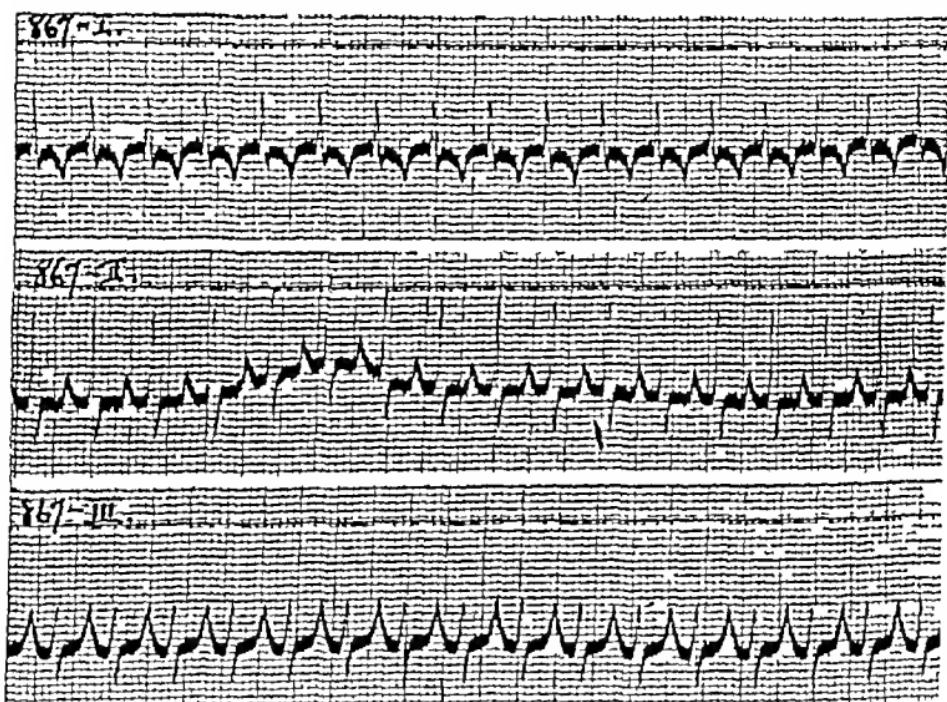


Fig. 230.—Electrocardiogram of Case II. P inverted in Lead I, summation of P and T in Lead III.

only a slight change—*i. e.*, from 190 to 180. The heart recovered promptly from the effect of the digitalis, and the child was discharged from the hospital nine weeks after admission. Her pulse ranged from 180 to 150, always regular, her temperature was normal, and she felt quite well, though rather easily fatigued.

For nine weeks after her discharge she was kept very quiet and had no subjective symptoms save an occasional dizzy spell. There was no evidence of cardiac incompetence during this period. The heart remained rapid—up to 190. Thirteen weeks

after discharge she returned to the dispensary. She had been running about a good deal, and became dyspneic on relatively slight exertion. Her temperature had remained normal. On examination, the heart showed a systolic murmur at the apex, transmitted to the axilla. The lungs showed moist râles at both bases.

Seven weeks later she returned to the dispensary for the last time. She had been kept very quiet, according to instructions,

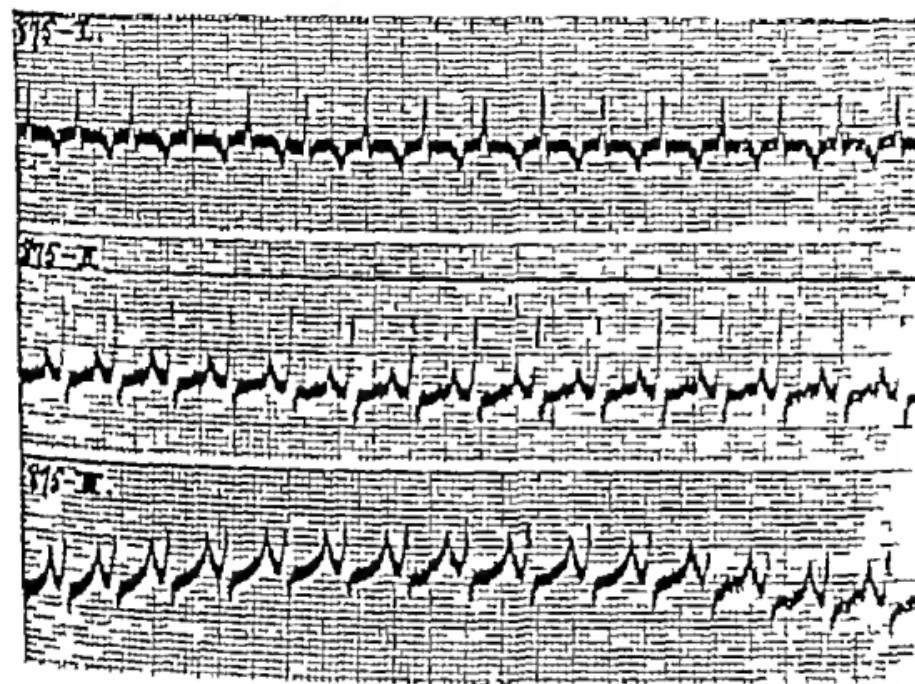


Fig. 231.—Electrocardiogram of Case II showing slight changes in initial complexes which might be interpreted as indicating progressive right heart hypertrophy.

but the rapid heart action, and the dyspnea on slight exertion had persisted. The left border of the heart was found 1 cm farther out than before, and the systolic murmur and râles at the bases of the lungs were still present. Three weeks after the last visit the child died of pneumonia complicated by heart disease, as stated by her physician.

Electrocardiograms, Fig. 230 (two days after admission to the hospital)—The rate is 190, the ventricle responding to every au

ricular impulse. There is some evidence of muscular preponderance of the left side of the heart. The striking features are the inverted P wave in Lead I, with the apparent summation of P and T where there is any evidence of a T wave. The P-R interval is either 0.14 or 0.18 second, according to the point chosen as the correct foot-point of P.

Figure 231, sixteen days after admission. She had received the equivalent of 0.3 gm. digitalis leaves in seven days. There is

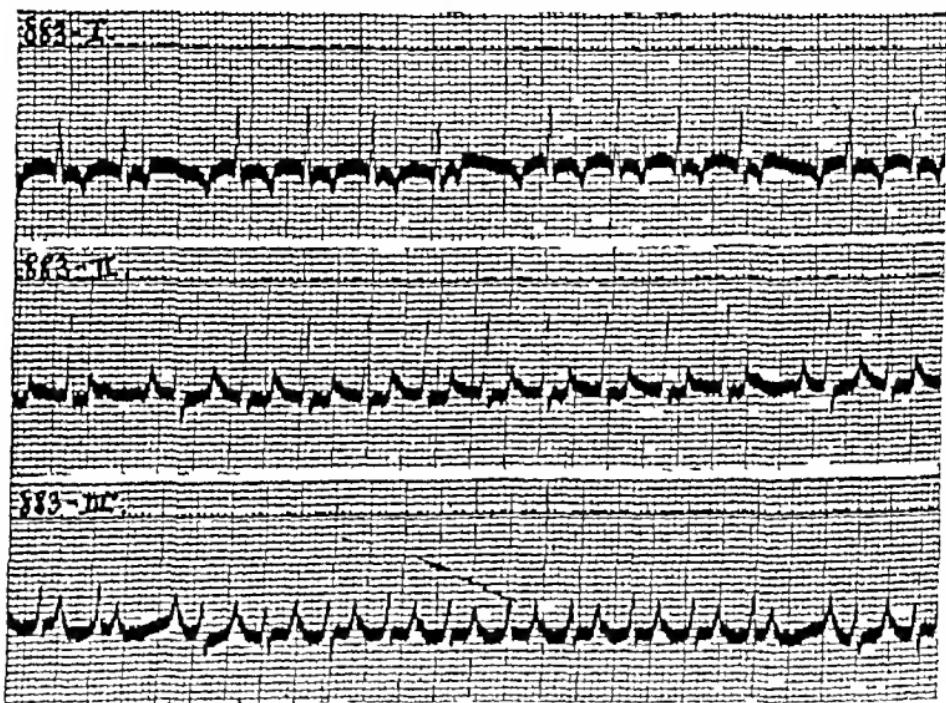


Fig. 232.—Electrocardiogram of Case II at the time of digitalis block, revealing the form of P when not modified by interference. The alterations in the initial complexes pointed out in Fig. 231 are still more marked here.

little if any significant change, unless the slight alteration in the relative amplitude of R and S in Leads I and III be taken to indicate a change in the muscle preponderance on the two sides of the heart. This, however, is a point of interest in connection with Figs. 230 and 232.

Figure 232, three weeks after admission. Partial heart-block due to digitalis, of which she had received about 1 gm., in terms of leaves, in two weeks. The auricular rate remains practically

unchanged (180), and the form of the auricular complex remains essentially as in the first record. There are changes in the relative size of the R and S waves, to a more marked degree than in Fig 231. It would be hazardous to interpret these as indicating a change in the relative muscular preponderance when the heart

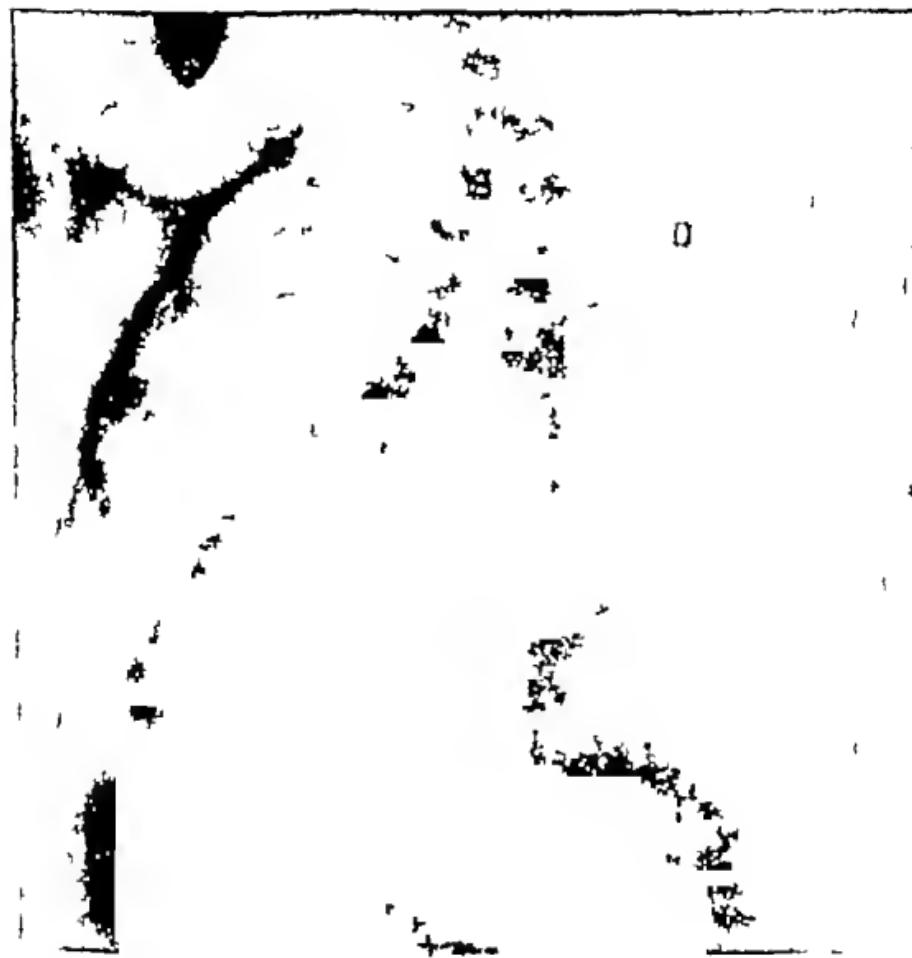


Fig 233.—Teleoentgenogram of Case II

is under the influence of digitalis. This record is of value in that the P wave is extricated from other complexes by reason of the block, thus enabling us to identify it (Fig 233).

Discussion.—In this case we are dealing with a tachycardia originating in the auricle and persisting in spite of all measures aimed to slow the heart. Both vagus pressure and digitalis,

which have been found effective in a certain number of these tachycardias, failed to affect the rate of the pacemaker, merely inducing depression of conduction at the junctional tissues. We have not as good evidence of an ectopic source of the rhythm as in Case I, since there is no change in the mechanism while under observation to serve as a basis for comparison. The constant high rate and the inverted P in Lead I are the points which favor the supposition that the rhythm is ectopic.

The matter of most importance in this case is the striking exhaustion of the cardiac reserve under the rapid rhythm. Whether we attribute this to fatigue due to so rapid a rate over a period of one year, or to primary disease of the myocardium, the condition was undoubtedly an important factor in lessening the child's chances against the terminal infection. The most reasonable supposition is that a primary myocardial lesion, due to some previous infection, determined the abnormal rhythm, and that myocardial exhaustion contributed toward the final outcome. In the one year of illness the heart probably expended the energy which should have been distributed over a period of two years or more.

Summary—Two cases of auricular tachycardia in children are described. One of them, by reason of its transitory character and the absence of clinical evidence of any other cardiac disorder, as well as by reason of the subsequent history, was, in all probability, functional. The other, because of its persistence in spite of all efforts to abolish it, and because of its association with definite evidence of cardiac injury, must certainly have had an organic basis. That the first one represents an ectopic rhythm there can be no doubt. That the rhythm in the second case also was ectopic seems highly probable.

CLINIC OF DR DANA W ATCHLEY

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RENAL DISEASE

Study of Renal Function Practical Application of the Kidney Activity to Clinical Material Prognosis Treatment. Presentation of Cases

March 1919

THERE are some diseases where we are fortunate in having a perfectly known cause that leads to a comparatively definite series of events, a discussion of typhoid fever, for example, should fix in the mind of the student a well-established picture of onset, course, and common variations. But certain other diseases are in no way made to order, for they show marked differences between individuals, and we are confined to the presentation of a point of view, a method of attack, diabetes is obviously such a disease. Nephritis has been too long taught as a disease of the first category, and many narrow groups have been presented, each with its supposed clinical picture. Practically, such grouping is impossible and is more often confusing than helpful to the student. Each case of renal disease is a distinct entity, and may be properly understood only after a certain minimum amount of material has been gathered. This material will suggest the prognosis and treatment in the case under study, but will fit the case into no neat pigeon hole. Before I discuss the accumulation and interpretation of the essential data in a case of renal disease brief mention should be made of the most rational classification that has been proposed, merely because the student demands some cataloguing, and a complete refusal may discourage interest.

In the absence of an etiologic basis, and when the pathologic and clinical pictures show uncertain coincidence, the most logical basis of classification must be physiologic. Widal first suggested the groups—salt retention, nitrogen retention, and mixed type. There is undoubtedly a clinical syndrome in which the predominant functional disturbance is an inability to excrete sodium chloride, and there is also another picture with nitrogen retention a marked feature, but the “pure” cases, if we may speak of them as such, are very infrequent in comparison with the cases where there is a combination of the two so-called types. In spite of this criticism Widal’s classification seems to be the best yet proposed, and is sufficient when hasty, casual description of cases is all that is required. It is to be recommended to those who desire a more particular terminology than the comprehensive term “renal disease.”

The accumulation of data in renal disease is so very important and there have been so many and varied contributions, that a rather detailed discussion may be useful in pointing out certain absolute essentials. Many complicated procedures necessary to the special investigator are valueless to the practical clinician.

In the first place, a careful anamnesis should include in the past history questions as to 1 Etiology—scarlet fever, sore throat, lead or mercury poisoning, evidence of protein sensitivity or asthma, this latter because of Longcope’s demonstration that experimental renal disease may be produced by continued injections of foreign protein. 2 Dyspnea on exertion, for its contribution to cardiac insufficiency. 3 Previous attacks of edema, or urinary disturbances, such as hematuria. 4 Previous urinary examinations, this an attempt to delimit the duration of the disease.

The present illness should point out if possible 1 A precipitating factor, such as acute infection or exposure to physical agents. 2 Headaches, if of the morning type so often seen in this disease. 3 Edema, its mode of onset and distribution. 4 Visual disturbances (easily confirmed by ophthalmoscopic examination). 5 Uremic manifestations, such as nausea, vomit-

ing, drowsiness, coma, twitching, or convulsions, are of very serious import. 6 Urinary abnormalities (a) Polyuria may mean loss of concentrating ability, (b) Oliguria, suggesting salt retention, if accompanying edema, (c) Hematuria, very strongly implying a real inflammatory process in the kidney 7 Dyspnea, this may be cardiac, may be due to hydrothorax as part of an anasarca, in some cases acidosis, or, finally, the type known as Cheyne-Stokes

The history, in the main, makes two contributions first and most important, the demonstration of unfavorable symptoms, and second, some idea as to the duration of the disease. I am purposely omitting nocturia because it is an unreliable symptom, influenced by many extraneous factors, and is, after all, entirely overruled by a study of the concentrating ability of the kidney Obviously a full history along other lines should be taken, the points I have mentioned are those never to be omitted in the examination of a patient suspected of renal disease

Physical examination may show no abnormality other than edema, this is the simplest and most hopeful case, but other patients have various abnormalities that indicate, in more or less degree, a severer disease Certain of these findings when present in renal disease are almost invariably serious, while others merely add to the list of bad signs. Of the first type are 1 Uremic manifestations—twitchings, coma, convulsions 2 Cheyne-Stokes breathing 3 Marked hypertension, 200 mm systolic and over 4 Acidosis, as demonstrated by Kussmahl breathing 5 Retinitis

The following are to be considered on the serious side, although not so consistently grave 1 Emaciation. 2 Enlarged heart. 3 Anemia. 4 Thickened arteries A red blood cell and hemoglobin estimation should always be done, for the observation of anemia in the mucous membranes is often very deceptive The number and degree of these physical findings present in a given case is the most valuable criterion of the severity of that patient's disease, entire absence of both groups with only edema present is the picture in pure salt retention, any of the first group with or without edema means nitrogen retention in

almost every instance. But a suggestive picture on physical examination should never permit omission of the functional studies.

The clinical examination of the urine should include 1. Careful measuring of the total daily output. Polyuria with a constant weight on a controlled intake may be an evidence that concentration is poor. Oliguria may be a very serious sign if nitrogen retention is present. 2. Specific gravity. Frequent observations under a variety of conditions, being very careful that the instrument used is accurate, the significance I shall discuss later. 3. Microscopic examination for blood, or, more significant, red blood-cell casts. The value of this observation is the light it throws upon the presence of a complicating inflammatory process. An occasional red cell is usually insignificant. 4. Albumin and casts are the least interesting findings when the presence of renal disease is once established. Much albumin may be found in the mildest cases, sometimes none in the most severe. A quantitative estimation of albumin is worthless for the clinician, even if the inaccuracies of the clinical methods for its determination were less gross.

Our fundamental knowledge of renal function is sadly deficient. We can say objectively that the kidney performs a number of necessary services for the human body. The mechanism of these things is still largely hypothetic. In fact, in some instances, we really do not know whether the kidney is an active or a passive agent. Most distressing of all is the poor co-ordination of what physiologic and anatomic facts we have at our disposal. It is clear, therefore, that a study of renal function is necessarily superficial. But much valuable clinical material may be gleaned by a few simple procedures. First of all, it is necessary to understand the various functions and the significance of each.

The important functions include the following. 1. The excretion of water, many factors influence water output, among them are fluid intake, temperature and humidity of outside air, exercise, water excretion by bowel. The use of water as a vehicle of excretion is very essential, for, as Cushny points out, there are

over forty six urinary constituents soluble in water, few of which are soluble in anything else

2 The excretion of sodium chloride the amount is dependent largely on the intake, and the kidney so acts as to maintain a fairly constant level of chlorids in the blood In this way the kidney is considered to be an important factor in preserving the necessary osmotic pressure of body fluids

3 The excretion of certain substances aids in maintaining the neutrality of body fluids (a) Acid phosphates are concentrated thirty times more in urine than in blood, a process of obvious value in getting rid of acid substances in the blood (b) Ammonia is excreted as a salt combined with undesirable acid radicals (c) Beta-oxybutyric acid is over two hundred times as concentrated in urine as in blood It is clear that a marked decrease in these functions would produce a degree of acidosis that the respiratory mechanism would not be able successfully to combat.

4 The excretion of waste products, largely nitrogenous, such as urea, creatinin, uric acid These substances are excreted normally as long as they are present in the blood There is no attempt to preserve a constant level as in sodium chloride

5 The excretion of foreign substances, such as poisons or dyes

6 The concentrating ability of the kidney should not logically be discussed with the preceding functions It is a method of work, not a result accomplished, but I have mentioned it here because a clinical summary of the renal function in any given case includes it with the others This function is a necessary safeguard to insure proper elimination when only small amounts of water are available to the kidney

Having in mind the rationale of the kidney's various activities, we next must learn their practical application to clinical material, and we may consider them in the same order, which, however, is not an order of importance

1 Water excretion, because of the many factors influencing renal water output, may be accurately studied only by constant observations of the body weight, an increase in weight meaning in general a holding up of fluids, a decrease, the reverse Fre-

quent weighing of an edematous patient is absolutely essential if the patient is well enough to permit.

2 Salt excretion may be determined by a very simple clinical examination of the urine. The materials for this test are inexpensive, the technic easy, there is no excuse for its omission by any practitioner. The specific holding up of salt in the uncomplicated edematous type represents often an excretion of less than 1 gm a day, while, although the more severe types without edema rarely excrete normal amounts, they seldom go below 1 gm. Of course, it must never be forgotten that the salt intake influences excretion markedly. During diuresis the salt output increases, and a relapse may often be predicted by a rapidly decreasing output of salt.

3 Evidences of acidosis, clinical and chemical, are proof of the decrease in this function, but the clinical value is slight because this condition always occurs in the very latest stage of the disease.

4 An impairment of the ability of the kidney to excrete nitrogenous waste-products is best studied by observing their accumulation in the blood. The simplest and most consistently satisfactory method is a determination of the amount of urea in the blood. Any man who has Wasserman reactions done on his patients can have this determination, drawing 10 c. c. of blood into a test-tube and sending it to the nearest chemical laboratory is all that is required to obtain this most valuable bit of information. No case of renal disease should be treated without at least one blood-urea determination. It is hard to give arbitrary figures on the upper level of normal. In general, on an average diet, an amount above 0.4 gm per liter of urea is suspicious of impaired function, and the blood urea should be watched carefully, if it drops rapidly, it is, of course, less serious than if it remains constantly high. Nitrogen retention is a serious finding, it makes prognosis considerably worse, it is invaluable in its suggestions for treatment, in short, it changes one's point of view entirely.

5 Study of phenolsuphonephthalein excretion is the clinical application of this function of the kidney. The test has no bear-

ing on any particular one of the other functions, nor is it the quantitative test that its percentage expression would indicate. In the summing up of a case it should play a secondary rôle, for example, a very low phthalein is one more bad sign in a severe case, but a figure of 30, when uremia, hypertension, and nitrogen retention are present, is deceptive. In other words, if the phthalein stands out as a single incompatible factor in the total picture, it should be discounted. In surgical conditions this test plays a much more important rôle.

6. Studies of the concentrating ability over a twenty four hour period by the so-called renal test meal are inconclusive unless they give positive evidence that the kidney can concentrate, for too many factors may influence water excretion during such a short time. Two observations of a high specific gravity are worth very much more than a dozen never going above 1010. A very low water intake is the ideal method of testing this function, but the greatest fixation at a low level occurs when nitrogen retention is present, which is a contraindication to restricted fluid. When conclusively demonstrated, an inability to concentrate indicates considerable involvement of the kidney. The relative importance of abnormalities in this function may be compared with the phthalein excretion, alone they are of less significance than when fitting into and confirming the rest of the picture.

One of the most essential as well as the most difficult tasks is the exclusion of extrarenal factors or their proper balancing with the renal, when they are present. Predominant among these factors is the cardiac. Each case should be carefully observed for evidences of cardiac insufficiency, and it should be remembered that passive congestion of the kidney may exaggerate a comparatively mild impairment of renal function. Many cases of cardiac insufficiency with hypertension are called chronic nephritis when there are no findings distinctively associated with renal disease (for hypertension is more frequently found in other circumstances), and when the very slight impairment of function first observed disappears as the heart compensates. There are many cases, however, where it is practically impossible to disentangle the various factors.

Prognosis—As this discussion has progressed the relative importance of certain evidences of grave import has been pointed out in the history, physical examination, and functional findings. The prognosis depends on the presence or absence of these factors. A patient whose history consists solely of edema, whose physical examination shows no other abnormality than edema, whose renal function is intact except for a specific inability to excrete salt and water—this patient has the best possible prognosis, he may live for years and be economically unaffected by his disease. Although, in any case of renal disease, the prognosis should be guarded, for the vagaries of the disease show themselves when least expected. Hematuria is significant because of its suggestion that an actual inflammatory process is present. The simple salt retention case I have just described may have present one or more of the serious findings in any and all combinations, and the edema itself may be absent. In any event, a prognostic analysis of the case means a gathering together of the grave signs, weighing their relative importance, and giving a common-sense opinion for the particular case at hand. In no disease is prognosis more difficult, but certainly more consistent correctness of prediction is possible with the point of view just described.

Treatment—An analysis of each case suggests the most rational treatment for that case, certain general principles obtaining.

1 Rest in bed is indicated (a) when edema is present, and should be continued for at least a week after its disappearance, (b) when hematuria is present, or (c) when the patient is too sick to be up.

2 Fluid intake should be limited to 1200 c c or below when there is retention of water as evidenced by edema. But if nitrogen is being retained, water must be given in larger amounts, at least 1800 c c if edema is present, and up to 2500 or 3000 c c if there be no edema. Inability to concentrate is a special indication for forcing fluids, because it means that the kidney requires much more water to excrete a given quantity of solids. If in the uncomplicated edematous cases limiting of fluid is unsuc-

cessful in producing a diuresis, forcing of fluids may, oddly enough, turn the tide in an occasional case.

3 The diet should always contain sufficient calories, there is never need for a starvation diet such as the Karrell, moreover, an attractive diet is quite as possible as an unattractive. For instance, use may be made of a large number of neutral vegetables containing no harmful substances, non-nitrogenous fruits are also available. Sodium chloride must be markedly restricted in the edematous cases that are holding up chlorids, and decreased in all cases empirically. This may be accomplished for practical purposes by the omission of salt when food is being prepared, by the use of fresh butter, and the avoiding of essentially salty foods, such as salt fish. Nitrogenous food materials, the proteins, are given in proportion to the ease with which they are handled by the kidney, if there is no increase in blood urea they may form a normal proportion of the diet, but a high blood urea means restriction of protein to about 35 gm a day. It must not be overlooked that eggs, fish, fowl, or beans are just as important as the so-called "red meats" when proteins are being considered. A patient who has once shown nitrogen retention should never be given more than 60 to 70 gm of protein a day. It is very helpful to follow the influence of a low protein diet on the amount of urea retained in the blood.

4 Drugs specifically irritative to the kidney, such as mercury, should not be used. Saline catharsis is worth trying in edema. There is no contraindication to the use of iron in the anemic case not at death's door. Diuretics have long been a bone of contention. Our present status is as follows. There is definite evidence, experimentally and clinically (Christian), that diuretics are usually not effective in renal edema, and may, indeed, be harmful, particularly if real inflammation of the kidneys be present. One should, therefore never use theocin or diuretin until he has been unsuccessful with all other means over a period of at least three weeks. At the end of that time one course may be tried and occasionally real success will attend the administration, but if no results are obtained, do not repeat the drug. Diuretics are not indicated in any case without edema.

5 Bleeding is a procedure of doubtful value in any case—both theoretically and practically—if, however, it is done on an anemic patient it is worse than useless. It is one of those therapeutic procedures of more value to the onlooker than to the patient.

6 Hot packs may be used in the edematous cases when there is no nitrogen retention, if, however, the nitrogen excretion be impaired, they are contraindicated. If the pack be successful and a large amount of fluid is excreted by the skin, that much less fluid is available to the kidney as a vehicle for the excretion of waste products, whose excretion by the skin is far less efficient than by the kidneys. It is possible to precipitate an attack of uremia in the nitrogen retention type of case by the use of hot packs.

7 In those cases where the prognosis is good, typically the salt retention type, a careful search for foci of infection should be made, and such as may be found removed. Tonsils and teeth are by far the most frequently found abnormalities.

Therapy is very discouraging because we can make no attack on the cause of the disease or diseases—for it is as yet unknown. All of the general principles I have mentioned are possible of exception. An open mind is absolutely necessary to a proper understanding and treatment of renal disease—the explosion of one's pet theories is inevitable if one continues to see cases. An attempt at rational analysis and treatment based on the few well-established facts at our disposal, should be our aim.

In conclusion I wish to present 3 cases.

CASE I—Man, aet twenty-seven years, admitted to the hospital complaining of swelling of his legs and ankles for two days.

Family History—Mother died at fifty-two of kidney trouble.

Past History—Hives at fifteen years, otherwise entirely negative.

Present Illness—Slight cough for one week, frontal headache three days ago, two days ago noticed that his feet were swollen, no other symptoms.

Physical Examination—Marked general edema, severe pyo-

Hg, definite acidosis 4 Blood urea = 3.31 gm per liter, a marked retention 5 Phthalein = 0 6 No positive evidence either way as to the concentrating ability of the kidney, but no high specific gravity observed

Course—Progressively down hill, with death ensuing in one month

Discussion—An apparently well man, whose history and physical examination showed two very grave signs—retinitis and marked hypertension—plus anemia and an enlarged heart, we would expect serious impairment of renal function, and, indeed, the urea is very high, phthalein very low, and an acidosis is present. All the findings agree on severe disease, and the course justifies that conclusion. Treatment was too late to be effective.

CASE III—Man, aet twenty-six, admitted to the hospital complaining of swelling of the face for ten days. Family history negative.

Past History—Has worked as a compositor for eight years, but has never had any symptoms of lead-poisoning. Bell's palsy eight years ago. No cardiac or renal symptoms.

Present Illness—Slight cough twelve or thirteen days ago. Swelling of face ten days ago, extending rapidly to whole body. No other symptoms.

Physical Examination—A general edema, carious teeth, large tonsils, heart enlarged to left, B-p 178/112, eye-grounds negative.

Blood—R b c = 4,350,000 Hb = 79 per cent. Smear = no stippling of red cells.

Urine—Amount consistently low—300 to 600 c.c. Sp. gravity, 1017. Albumin, very heavy trace. Micro, hyaline casts, few r b c, no lead.

Function—1 Water excretion very poor, increasing weight and edema. 2 Salt excretion less than 0.5 gm a day. 3 Blood CO₂ = 24.8 mm Hg, acidosis. 4 Blood urea = 1.56 gm. per liter. 5 Phthalein = 20. 6 No evidence on concentrating ability.

Course—The patient continued to retain salt and water. His blood urea went up and his phthalein excretion decreased. In two months he had become uremic, and died shortly afterward.

Discussion.—The history suggested simple salt retention. The physical examination offered no real evidence against this possibility except that the blood pressure was higher than one expects to find. Functional study showed a specific holding up of salt and water, and the phthalein excretion was not tremendously low—in other words, without a blood urea we would have given a good prognosis, treated him as Case I was treated, with restricted fluids, in short, been led quite astray. But the nitrogen retention immediately modifies our prognosis and our treatment. The course justifies the prognosis, if not the treatment.

CLINIC OF DR. EUGENE F. DUBOIS

CORNELL UNIVERSITY MEDICAL SCHOOL

THE BASAL METABOLISM AS A GUIDE IN THE DIAGNOSIS AND TREATMENT OF THYROID DISEASE¹

It is an easy matter to make a diagnosis in a typical case of hyperthyroidism presenting the cardinal symptoms of goiter, exophthalmos, tremor, and rapid pulse. Even a layman can tell what the trouble is at a glance if he has ever had a friend with Graves' disease. Typical cretinism can be diagnosed by anyone who has traveled in the Alps. In marked contrast to these extremes there is a large group in which it is most difficult to decide whether or not the patient is suffering from increased or decreased activity of the thyroid gland. These are the very cases in which a correct diagnosis is most important because of the good results that can be obtained by proper treatment.

Scores of tests have been presented as an aid in confirming the diagnosis of exophthalmic goiter. There is only one that stands out as a rational measure of the degree of hyperthyroidism, and that is the measurement of the basal metabolism. Since this is a matter of some complexity it may be well to discuss it before presenting the individual patients studied by this method.

The basal metabolism of a man is represented by the number of calories he produces in the morning hours, before breakfast while resting quietly in bed. Usually this is expressed in terms of calories per hour per square meter of body surface. The study of a large number of normal men and women has shown that the level of the basal metabolism varies with the age, sex,

¹ A series of cases in the metabolism ward of the Russell Sage Institute of Pathology in Bellevue Hospital presented to the students of Cornell University Medical College.

and surface area of the individual. Just why the metabolism should be proportional to the surface area is not clear, but the fact seems well established and is of great convenience in expressing the normal standards. Once having determined a man's heat production and having estimated his surface area from his height and weight, it is easy to compare the result with the normal standard for his age and state that his basal metabolism is such and such a percentage above or below the normal average. There are comparatively few normal individuals whose basal figures are more than 10 per cent. above or below the average. Practically all come within the 15 per cent limit¹.

A few years ago the measurement of the heat production was a difficult matter, attempted in only a few laboratories and clinics. At the present time there are half a dozen hospitals in this country where these tests are being made, especially on patients with exophthalmic goiter. In most places one of the simpler forms of apparatus is used, such as the Benedict universal respiration apparatus or the Tissot spirometer². These measure the oxygen consumption in periods of ten to fifteen minutes, allowing one to calculate the heat production by the method of indirect calorimetry. Much more complicated is the large respiration calorimeter, such as was used in the study of the patients here presented.

As might be expected, there are several diseases which cause an increase in the basal metabolism. Patients with high fever

¹ Lusk, *The Elements of the Science of Nutrition*, 3d ed., Phila., 1917, W. B. Saunders Co.

Gephart, F. C., and DuBois, E. F., *Clinical Calorimetry, Paper IV, The Determination of the Basal Metabolism and the Effect of Food*, *Arch. Int. Med.*, 1915, xv, 835.

DuBois, D., and DuBois, E. F., *Clin. Cal.*, Paper X, *A Formula to Estimate the Approximate Surface Area if Height and Weight be Known*, *Ibid.*, 1916, xvi, 863.

Aub, J. C., and DuBois, E. F., *Clin. Cal.*, Paper XIX, *The Basal Metabolism of Old Men*, *Ibid.*, 1917, xix, 823.

² Carpenter, T. M., *A Comparison of Methods for Determining the Respiratory Exchange of Man*, Carnegie Institution of Washington, Publication No. 216, Washington, 1915.

Benedict, F. G., *A Portable Respiration Apparatus for Clinical Use*, *Boston Med. and Surg. Jour.*, 1918, clxxviii, 667.

are 30 to 40 per cent. higher than normal. In severe cardiac and renal disease and anemia the results may be high, and in leukemias with very large numbers of white cells the heat production may be almost double the normal. Some patients with acromegaly give high results.

Fortunately, most of these conditions are easily diagnosed and are seldom confused with thyroid disease. It is in hyperthyroidism that we find the highest basal metabolism, and there is a striking parallelism between the severity of the disease and the level of the heat production. In very severe cases the increase is more than 75 per cent., in severe cases 50 to 75 per cent., and in moderately severe and mild cases less than 50 per cent. Conversely, in cretinism and myxedema the metabolism may be 20 to 40 per cent. below the normal average, figures lower than in any other condition except profound emaciation. In such cases of deficient thyroid secretion the metabolism can be raised by the proper administration of thyroid extract.

An increased basal metabolism is not pathognomonic of hyperthyroidism any more than sugar in the urine is pathognomonic of diabetes. In fact, there is a striking similarity between these leading manifestations of the two great diseases of metabolism—diabetes and hyperthyroidism. If a patient complains of polyuria, polydipsia, and loss of weight and strength in spite of an increased appetite, we suspect that there is some interference with the oxidation of the carbohydrates, and confirm the diagnosis by testing the urine for sugar. If another patient gives a history of mental irritability, rapid, throbbing heart action, warm, sweating skin, increased appetite, and loss of weight we recognize that these are the symptoms of increased metabolism, and if fortunate enough to have the means at our disposal we confirm the diagnosis of hyperthyroidism by measuring the heat production. In this manner it may be possible to diagnose "exophthalmic goiter" without waiting for the appearance of exophthalmos or goiter. In diabetes we gage the effect of treatment by determining how much carbohydrate can be oxidized. In hyperthyroidism we can follow the course of the disease by watching the fluctuations in the basal metabolism.

The first patient is Anna K.,¹ referred to the metabolism ward by Dr. John Rogers. She is a nurse, twenty-six years old, of a rather neurotic family. At the age of sixteen she had rheumatic fever, and since then has been high strung and easily frightened. For several years she has suffered from dyspnea and palpitation on exertion, and about a year and a half ago these symptoms grew worse, and she began to have severe headaches, marked sweating of the skin, excessive appetite, and thirst. Two months ago she was badly frightened in a runaway and all the symptoms increased in severity. Since then she has been taking a special thyroid "residue" prepared at the Loomis Laboratory. On April 4th both superior thyroid arteries were ligated by Dr. Rogers in this hospital.

On April 28th, at the time of the first calorimeter observation, she presented the picture of extreme hyperthyroidism except that there was only slight exophthalmos and no lagging of the lids on looking downward. The expression was tired and neurotic, the eyelids were puffy, and the voice weak. The frame was small and she was very thin, weighing only 98 pounds, which was 15 less than her usual weight. The thyroid gland was moderately enlarged, especially on the right side. The tongue and hands were tremulous, the skin moist and warm. The cardiac impulse was diffuse, maximum in the fifth space, 8.5 cm to the left of the midline. The action was regular, averaging 121 per minute.

The first hour that she was in the respiration calorimeter she produced 102.6 calories, the second hour, 101.5. According to her surface area this was 104 per cent above the normal average. This means that every square inch of her skin was giving off twice as much heat as in the case of a normal woman, so it is no wonder that it felt warm and moist. Her food requirement was double the normal, and her emaciation showed that the intake had not increased correspondingly.

On April 29th, the day after this observation, Dr. Rogers

¹ The details of histories and laboratory findings have been published in Clinical Calorimetry, 14th Paper, Metabolism in Exophthalmic Goiter, Arch. Int. Med., 1916, xvii, 915.

ligated the two inferior thyroid arteries. She stood the operation well, and on May 13th returned for another test in the calorimeter, feeling less nervous and more ambitious. This time she produced an average of 84.2 calories per hour, which was 71 per cent. above the normal average for a woman of her weight. The pulse-rate had fallen to 100 per minute.

This case illustrates several points. It shows that there can be an extreme degree of hyperthyroidism with only slight exophthalmos and moderate enlargement of the thyroid. It indicates an improvement following ligation of the arteries, but demonstrates that the gland can be markedly overactive even though all four thyroid arteries have been tied.

The second patient, Benny London, is at the other end of the scale of thyroid activity. He is a cretin of the type that used to be so common in some of the villages of Switzerland and Northern Italy. He has been at the Children's Hospital on Randall's Island for the last twenty years, and it is believed that he is about thirty-six years old. His mental and physical development corresponds to that of a small boy of seven. After twenty years of schooling he has finally learned how to write his name. Attempts to transplant sheep's thyroid into the abdominal wall and pelvis of the kidney failed to produce any beneficial results, and apparently caused the formation of a renal calculus. He has had numerous courses of treatment with Parke, Davis & Co thyroid extract, but on doses as small as $\frac{1}{2}$ to 1 grain three times a day develops tachycardia, weakness, and often syncope.

Benny is only 3 feet, $7\frac{1}{2}$ inches tall and he weighs 51 pounds. His face is broad, eyelids baggy, lips thick, nose broad, teeth widely spaced. No thyroid gland can be felt. There is a small umbilical hernia and there are pads of fat on either side of the neck and others just anterior to the axillæ. His genitals resemble those of a boy of seven and there are no male secondary sexual characteristics. The skin is very dry, harsh, inelastic, with fine branny desquamation. His Wassermann test is strongly positive, the urine contains much sterile pus, apparently due to the calculi in the pelvis of the kidneys. The temperature is slightly subnormal and the pulse 64 to 80.

Two calorimeter observations thirteen days apart show a basal metabolism 17 and 22 per cent. below the normal adult figure as based on his surface area. This means that he produces about half as much heat as a normal seven-year-old boy of his size because normal children show a very high basal metabolism.

After three days of treatment with thyroid extract, 3 grains a day, his metabolism returned almost exactly to the normal average for adults, but the temperature rose to 100° F., pulse to 100, and he felt so limp and looked so miserable that it was necessary to discontinue the extract.

No more popular patient has ever been in the ward than this little cretin. He is docile, uniformly good natured, full of fun, and blessed with a most contagious grin. At first his favorite occupation was hunting for bugs with a flash light, but he was disappointed by the negative results in Bellevue Hospital. He is a natural clown, and his humor is of a type that might well have been popular with the medieval monarchs who used these cretins as court jesters.

Our third patient, Max W., has been most unpopular in the ward because he teases his neighbors—the cretin and a pituitary patient of slow mentality. His excitable disposition and quick thoughts and actions show the driving force of too much thyroid secretion.

Max is a Rumanian Hebrew, forty years old, and for some time has been a storekeeper in this city. Two years before he came to the hospital he was refused life insurance because he weighed 190 pounds. A year before his first calorimeter observation he received news that his brother had been murdered and was much excited for a week. The next month he began to suffer from nervousness, loss of weight, and a severe, unproductive cough. He was sent to the mountains with a diagnosis of tuberculosis, but did not improve. It was then found that his Wassermann reaction was positive, and he was treated with mercury, still without improvement. Next he went to Mount Sinai Hospital, where they made a diagnosis of exophthalmic goiter and started medical treatment. He was discharged from this and one other hospital on account of quarrels with the nurses and

staff. He vehemently refuses surgical treatment because he saw a patient die after a thyroid operation. On the whole, he seems to have a poor opinion of the medical profession.

The patient is tall, but weighs only 136 pounds. His skin is warm and slightly cyanotic. The expression is angry and the eyes are staring, with slight protrusion. Although the upper lid covers about 2 mm. of the cornea, it does not follow the eye ball when he looks downward. There is some weakness of convergence, he winks but seldom, and the forehead wrinkles but slightly. His thyroid gland is soft and moderately enlarged, especially the right lobe. When admitted to Bellevue the cardiac apex was in the fifth space, 11 cm. to the left of the midline, and the action was rapid, 108 to 142, with a marked irregularity of the respiratory type. At the apex was a soft systolic murmur. The urine showed traces of sugar and the Wassermann was strongly positive.

On Feb 16th, five days after admission to the hospital, his basal metabolism was 75 per cent. above the average normal. Four days later it dropped to 60 per cent., but after a week rose to 65 per cent. above the average. On March 4th it had fallen again to 54 per cent. This shows the improvement that follows rest in bed without medication. After March 5th he was treated by Dr. Beebe, who gave him cytotoxic serum. The patient felt much better and did not even object to the local reactions, which at times were severe. The metabolism fell slightly, reaching 39 to 53 per cent. above the normal. A year later he returned to the ward, saying that he was much stronger and less excitable. He had gained 22 pounds in weight and had been able to work in his store. The goiter was a little smaller, the eye symptoms were unchanged, and the skin was still moist and warm. The heart was fibrillating and the apex was 13 3 cm. to the left of the midline. His metabolism was 45 per cent. above the average.

It has been stated that this patient's urine contained traces of sugar. He was by no means a diabetic, as is shown by the fact that his blood-sugar remained normal on a diet containing 200 to 300 grams of carbohydrate. Absolute proof of his ability to oxidize sugar was furnished by the calorimeter. After taking 100

grams of glucose his respiratory quotients showed that he was deriving 60 to 90 per cent. of his calories from carbohydrate combustion

This case illustrates that a patient suffering from hyperthyroidism can have his troubles ascribed first to tuberculosis and next to syphilis. If his doctor had examined his urine he might have been treated for diabetes also. The calorimeter indicated that rest in bed caused his basal metabolism to fall from 75 per cent above the normal average to 54 per cent. A year's treatment with serum was not able to cause a drop of more than 9 per cent.

Our fourth patient, Anna R., has been followed for two years. She was the first thyroid patient tested in the calorimeter. When first seen she was twenty-nine years old, unmarried, a saleswoman. Five years before this she had typhoid fever, and on recovery noticed a swelling of her neck. Since then she has suffered from tremor, palpitation, restlessness, and loss of weight, but has been able to work up to the time of admission. She thinks she has improved during the last two years, but says that her friends noticed that her eyes were staring about one year ago.

The patient is tall and thin, with distinct exophthalmos and von Graefe's and Stellwag's signs. The thyroid is moderately enlarged, there is a slight tremor of the tongue and hands. The heart is slightly enlarged, the pulse 84 to 88.

On May 10th her basal metabolism was 34 per cent above the normal average. On May 12th, under local anesthesia, the left superior and right inferior thyroid arteries were ligated and the tip of the left upper pole removed. On the 16th the other two thyroid arteries were ligated and the right upper pole removed. For fear that she might suffer from a sudden diminution of thyroid secretion she was given $\frac{1}{2}$ grain of thyroid extract every four hours until May 18th. On the 20th her metabolism was higher than before the operation, registering 55 per cent. above the average. Eight days later it was 52 per cent. This shows that an operation can increase the activity of the thyroid gland.

After leaving the hospital she spent a couple of months in the

country and gained weight and strength. She was then able to go back to work. A year after her operation she returned for a calorimeter observation. The thyroid was still enlarged, she was still weak and nervous, but her general condition was somewhat improved. Her basal metabolism was exactly the same as at the time of her first test.

A week later she was married and did well until her husband lost his job and she had to start work again in the store. When she reported at the metabolism ward two years after the first observation she was very thin, extremely nervous, and tremulous. Her skin was moist and the pulse small and weak. Unfortunately, it was not possible to measure her metabolism, but it was undoubtedly increased as a result of her efforts to support herself and her husband.

The fifth patient, Mrs. S., is in comfortable circumstances, but her breakdown was hastened by overwork, just as in the case of the saleswoman. She has always been energetic and athletic, but about five years ago developed an unusual degree of activity and restlessness. This led her to play tennis violently, and she won many cups in tournaments. She devoted herself to charities until it was evident that she was becoming nervous, irritable, and short of breath on exertion. A severe unproductive cough coupled with loss of weight made the family doctor suspect tuberculosis, and he sent her to a consultant. No diagnosis was made until the patient's mother in law suggested that the trouble was exophthalmic goiter, since the expression of the eyes reminded her of a man she knew who was suffering from this disease. A flood of light came to the family doctor, who promptly discovered a slight tremor, distinct tachycardia, puffiness of the eyelids, and unusual brilliancy in the eyes. On questioning, the patient admitted that she slept with very few blankets even on cold nights, and that her skin was always warm. In spite of treatment the condition grew worse and the exophthalmos became distinct, although there was never a goiterous swelling. Ligation of both superior thyroid arteries was performed by Dr. John Rogers, and during the next few years there was a slow improvement. The exophthalmos persisted, but the slight thick-

ening of the skin and some of the mental symptoms suggested that there was possibly an exhaustion of the gland with threatened myxedema. In order to see if thyroid extract were indicated Dr. Rogers advised a test to determine the level of the basal metabolism. The calorimeter showed that the heat production was 20 per cent above the normal level and that the gland was still in the stage of hypersecretion.

The sixth and last patient, Peter N., was a puzzler. He was considered to be a case of atypical exophthalmic goiter and was referred to the metabolism ward to see if an operation were advisable. He was twenty-three years old and 6 feet 2 inches in height. His father was 6 feet 4 inches. The patient grew fast between the ages of fifteen and eighteen and became weak and nervous, losing his temper easily. He suffered from headaches two or three times a week, and about a year ago his hands grew so tremulous that he had to give up his work as mechanic. Shortly after this he went to the Massachusetts General Hospital, where his respiratory metabolism was studied by Dr. J. H. Means and found to be within normal limits. The superior thyroid arteries were ligated by the surgeons, and since then the headaches have been rather less frequent, but he is not much stronger and he only sleeps two or three hours at night.

The young man is tall and thin, but there is no suggestion of acromegaly in his appearance and the x-ray shows that the sella turcica is normal in contour. His expression is anxious, but there are no eye symptoms. The thyroid is soft and slightly enlarged. The hands and feet are sweating and there is marked dermatographia. Cardiac dulness extends 12 cm. to the left of the mid-line and the electrocardiogram shows a slight respiratory arrhythmia. The pulse-rate is only 71. There is a trace of sugar in the urine.

One observation in the calorimeter showed that his metabolism was 2 per cent below the average, in other words, absolutely normal. This, together with the test made a year previously by Dr. Means, rules out hyperthyroidism. In the light of our present knowledge we might suspect that this is a case of neurocirculatory asthenia.

As we review the preceding cases from the purely clinical aspect we are struck by the importance of symptoms outside the classical group of exophthalmos, goiter, tremor, and tachycardia. As Plummer has pointed out, the earliest symptoms are cerebral stimulation, vasomotor disturbances of the skin, tremor, mental irritability, tachycardia, and loss of strength. In two of the cases we have noted that there was an irritative cough, which led the physician to suspect tuberculosis. Before there is distinct exophthalmos there is usually a peculiar look of the eyes, an unusual brilliance, a slightly startled, angry expression. There is also an impulsiveness of manner and distinct restlessness of disposition. Often the patients complain that they cannot get along with people and especially with their own families.

Under ordinary conditions a man loses about three-quarters of the heat he produces by radiation and conduction from the surface of his body. About one-quarter is lost by the evaporation of water from skin and lungs. Therefore the increased heat production in hyperthyroidism manifests itself in a warm skin which breaks out into sweat on slight provocation. The patient even while at rest feels warm, just as if he were exercising. He needs but little covering at night. To supply all this extra heat it is necessary for him to eat large quantities of food.

We have seen that in the cases here presented the basal metabolism has been an accurate guide as to the degree of thyroid activity. The question naturally arises as to whether or not this holds good for all cases. Past experience has shown that there are few pathognomonic signs in medicine. Particularly in diseases of the ductless glands scores of tests have been discarded as worthless. It will take several hundred metabolism experiments with patients carefully standardized from the clinical viewpoint before one can say that the basal metabolism is an absolutely accurate guide in the diagnosis and treatment of thyroid disease. We can, however, at the present time say that it is the best guide, much safer than any other one sign or symptom. It is a better guide in doubtful cases than the opinion of nine out of ten consultants.

The test has a firm physiologic and pathologic basis. It is purely objective, and cannot be influenced by the hopes of the patient or his physician. Since the phenomenon was first discovered by Friedreich Müller in 1893 and confirmed two years later by Magnus-Levy, its significance has not been disputed, although it has been grossly ignored. In Germany the level of the metabolism has been used in a few experiments as a method of testing different forms of treatment. In this country, shortly before the war, a large number of observations were being made in various clinics. In recent publications by Means and Aub,¹ White and Aub,² and by Peabody, Wearn, and Tompkins³ it seems to be accepted as a standard of thyroid activity. Peabody has shown that a large proportion of the cases referred to the Army General Hospital at Lakewood as hyperthyroidism were incorrectly diagnosed.

Granting the value of the test, is it practicable? Calorimeters are out of the question except in a few laboratories. On the other hand, the simpler forms of apparatus for determining the basal metabolism are comparatively reasonable in cost and fairly easy to operate. They are much less expensive than an electrocardiogram and the technic is not so difficult. Still there are many pitfalls waiting for the careless investigator, and it is advisable that any man who starts to measure the total metabolism should begin with a series of normal controls.

"But why," says the practitioner, "should I, who have seen hundreds of cases, go to all this trouble when I can perfectly well tell from the clinical aspect whether or not the metabolism is increased?" Such guessing is very common, quite fascinating, and not risky for one's reputation unless the results are checked. On the other hand, those clinicians who have checked up their guesses in two or three hundred experiments realize the fre-

¹ Means, J. H., and Aub, J. C., *A Study of Exophthalmic Goiter from the Point of View of the Basal Metabolism*, *Jour Amer Med Assoc.*, 1917, 69, 33.

² White, P. D., and Aub, J. C., *The Electrocardiogram in Thyroid Disease*, *Arch. Int. Med.*, 1918, 22, 766.

³ Peabody, F. W., Wearn, J. T., and Tompkins, Edna H., *The Basal Metabolism in Cases of the "Irritable Heart of Soldiers,"* *Medical Clinics of North America*, September, 1918, U. S. Army Number.

quency of their errors, especially in atypical cases, and are not at all surprised if they do not come within 40 per cent of the true figure.

The diagnostician who interprets the basal metabolism intelligently will be able to determine the degree of thyroid activity. The surgeon who operates on thyroid patients will find that the basal metabolism is of great help in following his results. He will also find that in many cases a fifteen minute test with the respiration apparatus will save the patient a needless operation.

CLINIC OF DR. WILLY MEYER

LENOX HILL HOSPITAL

ADVANCED PULMONARY TUBERCULOSIS,
A BORDERLAND DISEASE

Presentation of a Patient Operated Upon with Subsequent
Results.

GENTLEMEN

For many years it has been my pleasure to select from my personal practical experience, for presentation before you, cases which are types of the borderland diseases

It is well known to you that in the course of the last decade many patients afflicted with all the well known diseases of the gastro-intestinal tract, with its contributory adjacent glands, the liver and gall-bladder, and pancreas, as well as the spleen, no less than many affections of the central nervous system and urinary system, have become borderland cases. At any time they may pass from the domain of the medical man across the borderline into surgery. The "when" depends on the inclination of the attending physician, or one might perhaps better say on his "conservatism". There are still some of our colleagues, thoroughly educated and learned men, who abhor the entrance of the surgeon into their fields, who cling to their chemical and physical therapeutics to the very last, and often assign even the cancerous affections of the organs mentioned above to the surgeon for operation when it is too late. To my mind, the standing of the modern physician can and must be estimated by his mental and energetic grasp of what medical science calls "borderland" today, by his readiness and the timely call of his conscience to realize that in the given case his personal, no matter how careful,

attendance can be superseded by a scientific operator to the advantage of his client

One of the latest chapters added to the borderland list is that of advanced pulmonary tuberculosis

It is generally recognized today that artificial pneumothorax (Forlanini-Murphy) is of great assistance in the treatment of pulmonary tuberculosis. Carefully supervised treatment by the internist or specialist, in combination with proper hygienic measures, are the necessary additions. No doubt many patients suffering from tuberculosis of the lung get well under such treatment, others are so improved as to lead a satisfactory existence for many years.

But what, if adhesions have united the pulmonary to the costal pleura, if the needle again and again fails to find a place in which the nitrogen or pure air can be blown in successfully, if also all the other measures of scientific régime do not bring improvement? If no other treatment were at hand, or resorted to, all these patients would be doomed, they would surely die within a brief space of time.

It is in these patients and at this stage that modern operative surgery steps in.

I. BRAUER-FRIEDRICH METHOD

Friedrich, of Marburg, on the initiative of Brauer, then the internist of the same university, took up this question in 1908. They reasoned that the required collapse, compression, and artificially putting at rest of the diseased lung could be accomplished by attacking the skeleton of the chest, if the presence of far-reaching adhesions between the two pleural leaves prevented the nitrogen or air insufflation into the pleural sac. They argued that resection of the tenth to the second or first rib, inclusive, with subsequent artificial mechanical compression of the side of the chest from without must in such cases accomplish, in part at least, the same final result as the Forlanini-Murphy method was doing in more favorable cases with non-adherent pleurae. Friedrich thereupon cut out the ribs mentioned after having gained the necessary access to the bony thoracic wall.

by means of Schede's incision. The work was done in one sitting under a superficial morphin-chloroform anesthesia, and was termed "extrapleural thoracoplasty."

Friedrich addressed the American Surgical Association on this subject at its meeting in Philadelphia in June, 1909. In 1911 he was able to report on 28 patients. There were 8 deaths (28.5 per cent.) and 20 recoveries from the operation, of the latter, 16 were much improved, 3 improved, together = 68 per cent., and 1 not improved. All the patients had been desperately ill. Nevertheless 68 per cent., — two-thirds, were improved by the operation.

II. SAUERBRUCH'S METHOD

For weak and reduced individuals, as represented by the majority of these patients, the Brauer-Friedrich procedure represents a serious undertaking. Friedrich's genial first assistant, F. Sauerbruch, therefore tried to modify and simplify it by operating in stages and under regional plus local novacain anesthesia. In order to avoid the threatening aspiration-pneumonia of the lower lobe from a purulent cavity in the upper lobe, he proposed, first resecting the tenth to sixth ribs inclusive and compressing the lung with a pad and elastic straps, and then, two or three weeks later, remove sufficiently large pieces of the fifth to the second or first ribs. He also showed that only the posterior half of Schede's incision is required for an exposure of a sufficient length of the ribs for resection (hook incision).

The latest results published by Sauerbruch in 1913 are remarkable. Of 113 patients treated with extrapleural thoracoplasty he operated upon 77 for advanced pulmonary tuberculosis. Of these, 17 were cured, 20 greatly improved, 19 improved, together 56 = three-quarters = 73.7 per cent., 4 remained unchanged = 5.2 per cent., 3 got worse = 4 per cent., 2 died soon after the operation (2.6 per cent.), 11 died of tuberculosis later on (14.5 per cent.), together death rate = 17.1 per cent. All the cases were carefully selected by specialists in tuberculosis, and the majority of them came down for the operation to the University Clinic at Zurich from Davos in Switzerland,

and returned to the higher altitude after the operative recovery "Unilateral affection" was the watchword, though slight involvement of the opposite lung did not definitely exclude the patient from the operation. Recent or progressing tubercular affection of the other lung and tuberculosis of the gastro-intestinal tract with diarrhea were considered a contraindication.

Sauerbruch succeeded in doing the operation with his trained set of permanent assistants, from the first incision to finishing the last skin suture, in nineteen minutes, a brilliant illustration of the effect of "crew-work," as it was witnessed by members of the Southern Surgical Club who had gone to Zurich on their laid-out itinerary through the United States and Europe in the early summer of 1914.

Gradually an effort was made to do the resection of the tenth to the second rib in the same séance in stronger patients. If it was seen later on that additional compression of the lung was required to accomplish the desired result, the resection of the first rib from the front, also resection of the phrenic nerve at the neck (phrenicotomy) for permanent paralysation of the half of the diaphragm on the diseased side, and Tuffier's operation, "apicolysis," with insertion of a plombe of fat or paraffin, were added. The latter material was pushed into the space which had been artificially produced by separating the costal plus the adherent pulmonal pleura of the apex from the endothoracic fascia, and thus working down gradually, collapsing the apex. Resection of a short piece of the second or third rib in the preaxillary line with division of the posterior periosteal envelope of the rib started the operation and exposed the costal pleura.

The immediate result of this far-reaching mechanical collapse and compression of the lung is proliferation of connective tissue, gradual carification of the lung, and reduction in size of former cavities. A pronounced chronic hyperemia sets in within the collapsed lung, according to Cloetta's careful investigations, which no doubt works in the sense of Bier's hyperemia, substituting the tuberculous infiltration with permanent scar tissue.

Hand in hand with this local organic improvement the

subsequent condition of the patient points to a steady change for the better. Cough with sputum becomes reduced or disappears, fever and night-sweats stop, tubercle bacilli can no longer be found, and the weight increases.

III. WILMS' METHOD

Wilms, of Heidelberg, did not fear the possible pneumonia in the lower lobe by aspiration, in consequence of multiple rib resection, in the presence of a cavity in the superior lobe. He favored the so-called "columnar resection" of the upper seven or nine ribs, anteriorly and posteriorly, in one sitting. The intermediate portion of the ribs left in place was used for more effective compression. He too worked under regional and local anesthesia. In 12 cases published there was no mortality.

All patients were much improved.

It is most interesting that the operative results of these three authors, who published the greatest series of such cases, are almost identical. All three succeeded in more or less arresting the disease in from 57 to 73 per cent. of their patients, that is to say, as many as 57 to 73 people out of 100 were either cured or much improved or improved. Certainly a remarkable record.

Personally, I have done the extrathoracic thoracoplasty seven times on advanced bronchiectasis in the course of the last ten years.

For this class of cases the operation represents one of the many so-called conservative operations which have been proposed for the surgical treatment of these unfortunates. It was the late Professor Friedrich, of Marburg, who persuaded me to proceed rather conservatively in the surgical treatment of patients suffering from advanced bronchiectasis, at the time of his visit to this country in 1909. He was of the opinion that every surgeon interested in thoracic surgery should make it his duty not to resort to lobectomy at once, but try less dangerous operations first. He feared that the youngest child of operative surgery, thoracic surgery, would not gain the confidence of our medical confrères and of the public if too many deaths were reported on the basis of too aggressive surgery. He there-

fore preached conservatism. Personally I followed him, and today look back on a series of conservative operations carried out on bronchiectatic patients, partially with regret and partially with satisfaction. With regret, because I failed to cure a number of advanced cases with bronchiectasis who might have had a better chance to live with properly performed lobectomy, and with satisfaction, because I succeeded with patience and perseverance in saving a few much-reduced patients from this terrible affliction who would most likely have succumbed to more radical work. There can be no doubt in my mind that exceptionally even somewhat advanced bronchiectatics can be *cured* by operations less serious than lobectomy.

At the present moment most careful individualization to establish the indications for operation is required in this class of cases. There can, however, be no discussion that in the advanced stage of bronchiectasis *only* the extirpation of the diseased lung lobe or lobes can bring permanent relief.

Looking back, a further source of satisfaction derived from my prolonged conservative operative work in this branch of surgery has been that I have made early acquaintance with extra-pleural thoracoplasty under regional and local anesthesia. As stated before, I performed it seven times on these patients, a few times resecting the upper ribs from the tenth to the first inclusive, at one sitting, and conversed with the non-narcotized patient during the entire course of the operation.

Now, gentlemen, anybody might very properly ask me: How did it come about that this operation was performed on 7 cases of bronchiectasis and never before on a consumptive in the last ten years? The answer is that I was absolutely prevented from carrying out my intention to do so, in spite of frequent applications, by the board of directors of the various institutions with which I am connected. Not that these gentlemen were opposed to the operation, but because they were prevented from permitting the admission of patients with active tubercle bacilli in their sputum to our hospitals on the basis of an order of the proper health authorities. A patient with active tubercle bacilli in his sputum cannot be placed next to a patient suffer-

ing from a different disease in our public wards. In our private hospitals connected with the general hospitals it was rightly feared by the managers that the inmates would resent the presence of an advanced coughing and expectorating tubercular patient on the same floor. Thanks to the remarkable education of the public in matters tuberculous this certainly was to be feared.

In the case of the patient to be presented today, a thorough discussion of the subject before the house committee of the Lenox Hill Hospital resulted in permission to have such a case admitted to the isolation house. Fortunately, the courtesy of the superintendent permitted the use of an isolated room on the top floor of the main hospital.

The patient here before you, L. S., twenty six years of age, was admitted to the Lenox Hill Hospital in November, 1918, having been sick with tuberculosis of the left lung since 1912. He had been residing since then, compiling chronologically, in Saranac Lake, Colorado, New Mexico, New York and again in Saranac Lake. Tubercl bacilli were first found in 1914. The establishment of an artificial pneumothorax was impossible on account of wide adhesions, and he was sent to me by Dr. Baldwin and Dr. Trembley of Saranac Lake for operation.

The patient appeared very delicate, was short of breath on the slightest exertion, and had a rapid pulse. Every few weeks his temperature, following a slight chill, rose to 103° F. and more, and returned to normal gradually. The left lung showed clinically all the signs of far reaching tuberculous infiltration with multiple cavity formation. The quantity of sputum equalled 60 c.c. in twenty four hours. The right lung was but slightly affected.

It was decided to take the safest course and do, for the rib resection, a two-stage operation.

On November 18th the first stage was done, with regional and local novocain and adrenalin anesthesia. After infiltration of the desired line of incision the Kappis method was carried out first, 2 c.c. of the anesthetic solution ($\frac{1}{2}$ per cent. novocain) being deposited after striking the rib with a needle,

and 8 cc around the posterior branch of the thoracic nerves near the spine. Then the Schumacher method, also injection of novocain in weak percentage, was added, 5 c.c. of the same fluid being deposited at and below the angle of the tenth to the sixth left rib, 150 c.c. of the $\frac{1}{2}$ per cent in all were used. After some temporizing, during which time the patient was finally prepared for operation, the lower three-quarters of the typical posterior hook incision were painlessly carried out, the ribs sufficiently exposed after pulling the scapula aside and a piece of bone, 10 to 15 cm long, from those ribs excised, well beyond the angle of the ribs. With two long split drainage-tubes in place, the wound was sutured in layers with a continuous stitch, and the dressing held in place by broad zinc adhesive plaster which also fixed the arm.

On the third day the first dressing was changed, when it was seen that the wound had healed by primary union. Two weeks later the patient was out of bed, improved in every respect. I made provision to begin at once with elastic compression by means of a pad, pulled by elastic straps to a loop surrounding shoulder- and hip-joint of the opposite side.

On December 5th, seventeen days after the first operation, it was considered wise to add the second stage, resection of the fifth to the second rib inclusive, again avoiding general anesthesia. Only a few drops of anesthol, more for quieting the patient's anxiety, were required once, for a short period. In order to expose a sufficient length of the upper ribs it became necessary, at the second stage, to incise the entire length of the first cut (Fig 234), a rather pitiful procedure after primary union. The regret felt suggested trying to do the required resection in one stage if possible on somewhat preserved patients.

A flat cavity filled with serosanguinolent fluid was encountered near the spine. The latter evidently was aseptic, patient having had perfectly normal temperature continuously. The stumps of the ribs resected at the first operation were found to be covered with new, glistening, white connective tissue of remarkable thickness. Again drainage and dressing were applied as before. The patient was out of bed on the sixteenth

day. Compression with large and small pads day and night was arranged for.

Today the patient is greatly improved. His sputum is reduced and coughing spells are rare, the dyspnea is decreased,



Fig. 234

he has gained in weight. The tubercle bacilli disappeared after the first operation and have never again been found up to date.

Through the courtesy of Dr. Baldwin, of Saranac Lake, the patient was allowed to return to that place. From there he writes to me under date of March 2d that he feels fine and strong and has full use of his left arm. His rectal temperature

has never trespassed 100° F, pulse is between 70 and 90 at rest and after exercise between 80 and 108, has had a severe coughing spell only on two days within six weeks, sputum is reduced one-half or more in quantity, he is able to walk many blocks without fatigue, his weight is gradually increasing

Considering the fact that this patient was hopelessly lost without operation, his present condition is a source of continuous satisfaction

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